A Dissertation on

"UTILITY OF LEFT BUNDLE BRANCH BLOCK AS THE DIAGNOSTIC CRITERION FOR MYOCARDIAL INFARCTION IN A HEMODYNAMICALLY STABLE PATIENT".

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for the award of the degree of

M.D.GENERAL MEDICINE

BRANCH – I

COIMBATORE MEDICAL COLLEGE AND HOSPITAL

COIMBATORE

APRIL – 2016

CERTIFICATE

This is to certify that this dissertation in "**UTILITY OF LEFT BUNDLE BRANCH BLOCK AS THE DIAGNOSTIC CRITERION FOR MYOCARDIAL INFARCTION IN A HEMODYNAMICALLY STABLE PATIENT**" was a work done by **Dr. ASHOK S.**, under my guidance during the academic year 2013 – 2016.

This has been submitted in partial fulfilment of the award of MD degree in General Medicine (Branch - I) by The Tamilnadu Dr. MGR Medical University, Chennai – 600 032.

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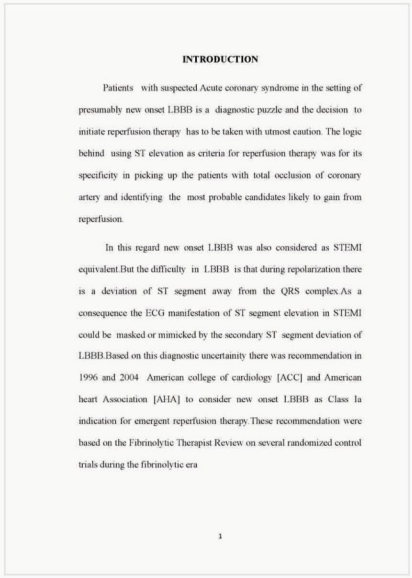

INTRODUCTION

Patients with suspected Acute coronary syndrome in the setting of presumably new onset LBBB is a diagnostic puzzle and the decision to initiate reperfusion therapy has to be taken with utmost caution. The logic behind using ST elevation as criteria for reperfusion therapy was for its specificity in picking up the patients with total occlusion of coronary artery and identifying the most probable candidates likely to gain from reperfusion.

In this regard new onset LBBB was also considered as STEMI equivalent.But the difficulty in LBBB is that during repolarization there is a deviation of ST segment away from the QRS complex.As a consequence the ECG manifestation of ST segment elevation in STEMI could be masked or mimicked by the secondary ST segment deviation of LBBB.Based on this diagnostic uncertainity there was recommendation in 1996 and 2004 American college of cardiology [ACC] and American heart Association [AHA] to consider new onset LBBB as Class Ia indication for emergent reperfusion therapy.These recommendation were based on the Fibrinolytic Therapist Review on several randomized control trials during the fibrinolytic era

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I **Dr. ASHOK .S** hereby declare that this dissertation entitled "**UTILITY OF LEFT BUNDLE BRANCH BLOCK AS THE DIAGNOSTIC CRITERION FOR MYOCARDIAL INFARCTION IN A HEMODYNAMICALLY STABLE PATIENT**" is a bonafide and genuine research work carried out by me under the guidance of **Prof.Dr.KUMAR NATARAJAN M.D.,** Department of General Medicine, Coimbatore Medical College and Hospital, Coimbatore, in partial fulfilment of regulations for the award of M.D. Degree in General Medicine to be held in April 2016.

This dissertation has not been submitted by me on any previous occasions to any university for the award of any degree.

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LIST OF ABBREVIATIONS

LBBB	–	LEFT BUNDLE BRANCH BLOCK
AMI	–	ACUTE MYOCARDIAL INFARCTION
MI	-	MYOCARDIAL INFARCTION
STEMI	–	ST ELEVATION MYOCARDIAL INFARCTION
CAHD	-	CORONARY ARTERY HEART DISEASE
SHT	-	SYSTEMIC HYPERTENSION
DM	-	DIABETES MELLITUS
TROP T	–	TROPONIN T
ECG	-	ELECTROCARDIOGRAM
CKMB	-	CREATINE KINASE MB
CABG	-	CORONARY ARTERY BYPASS GRAFTING
PTCA	-	PERCUTANEOUS TRANSLUMINAL CORONARY ANGIOPLASTY

INTRODUCTION

Patients with suspected Acute coronary syndrome in the setting of presumably new onset LBBB is a diagnostic puzzle and the decision to initiate reperfusion therapy has to be taken with utmost caution. The logic behind using ST elevation as criteria for reperfusion therapy was for its specificity in picking up the patients with total occlusion of coronary artery and identifying the most probable candidates likely to gain from reperfusion.

In this regard new onset LBBB was also considered as STEMI equivalent.But the difficulty in LBBB is that during repolarization there is a deviation of ST segment away from the QRS complex.As a consequence the ECG manifestation of ST segment elevation in STEMI could be masked or mimicked by the secondary ST segment deviation of LBBB.Based on this diagnostic uncertainity there was recommendation in 1996 and 2004 American college of cardiology [ACC] and American heart Association [AHA] to consider new onset LBBB as Class Ia indication for emergent reperfusion therapy.These recommendation were based on the Fibrinolytic Therapist Review on several randomized control trials during the fibrinolytic era

But Later studies using angiography concluded there is no documented coronary occlusion angiographically in majority of the people with new onset LBBB.Now there is a paradigm shift in the management of the patient with new onset LBBB and the new 2013 STEMI guidelines byAmerican College of cardiology and American Heart Association has removed the previous recommendations that the new onset LBBB should be treated as STEMI equivalents

My studyis to find the incidence of Acute Myocardial Infarction in patients with potential ischemic symptoms in relation to the presence of old or new onset LBBB and to analyze whether new onset LBBB predicts increased likelihood of Acute MI by monitoring with serial Troponin T and echocardiography.

Aim

To study the relevance of LBBB as diagnostic criteria for Acute myocardial Infarction

Objectives

To study the incidence of Acute Myocardial Infarction in patients with potential ischemic symptoms in relation to the presence of old or new onset LBBB

To analyze whether new onset LBBB predicts increased likelihood of Acute MI

REVIEW OF LITERATURE

Myocardial Infarction is one of the most challenging clinical conditions in cardiology but also the most gratifying when treated promptly and appropriately. Recent advances in the management of MI have shown the prompt treatment can save lives and restore normal cardiac function.. Delay or sub optimal management at any stage can affect outcomes.

The National Commission on Macro-economics and Health has estimated the evolving epidemic of CAHD in India - 60 million patients by the year 2015 with an annual mortality three million. A good number of this will be caused due to acute Myocardial Infarction or its delayed consequences. Even though Europe and United states have substantially fewer STEMI, the national bodies have formulated appropriate guidelines and there are audits and attempts to encourage and monitor adherence. Unfortunately there is no national guideline or policy in India

Rapid and prompt diagnosis of MI is extremely important in initiating reperfusion therapy . For most of the patients presenting to emergency department with chest pain, ECG still is the first foremost and most important diagnostic tool in identifying the patient for

reperfusion therapy. However patients presenting with concomitant left bundle branch the electrical manifestation of Myocardial Infarction may be masked.

Left bundle branch block presents a dilemma for many clinicians in the evaluation of chest pain (or other signs and symptoms of ACS).

- There is a pervasive myth that it's impossible to diagnose acute STEMI in the presence of left bundle branch block
- Left bundle branch block causes a secondary ST/T-wave abnormality that makes the diagnosis of acute STEMI more difficult
- "New" left bundle branch block was long considered to be a STEMI equivalent (many still believe this to be the case)
- Historically there has been a high rate of "false positive" cardiac cath lab activations based on the presence of left bundle branch block
- The diagnostic criteria to identify acute myocardial infarction in the presence of left bundle branch block (Sgarbossa's criteria) was originally based on a rise and fall of cardiac biomarkers, not angiography (in other words it combined STEMI and NSTEMI)

For these reasons, when it comes to field activation of the cardiac cath lab, most EMS protocols exclude patients with left bundle branch block or paced rhythm.

“It is impossible to diagnose STEMI in the presence of left bundle branch block”

The myth is well rooted in the medical literature.

“It is well recognized that the electrocardiographic diagnosis of myocardial infarction in the presence of complete left bundle branch block is in most instances difficult and frequently impossible.” Int J Cardiol. 1983;2(5-6):521-9

“It is common knowledge that the ECG diagnosis of completed myocardial infarction in the presence of left bundle branch block (LBBB) is extremely difficult and often impossible.” – Ann Emerg Med. 1995 Jul;26(1)69-82

This was considered to be a STEMI equivalent as recently as the 2010 AHA ECC Guidelines! And 2012 European guidelines

. “The 2010 ACC/AHA STEMI guidelines recommended emergent reperfusion therapy including fibrinolytics or primary percutaneous coronary intervention (PCI) in patients with symptoms compatible with

STEMI and new or presumably new LBBB if these symptoms arose in the prior 12 hours (Class I Indication)"

"The 2012 European Society of Cardiology STEMI guidelines also recommend early PCI or fibrinolytics in patients with a clinical presentation of STEMI and new or presumably new LBBB if symptoms arose in the prior 12 hours (Class Ia Indication)"

The 2013 ACC/AHA STEMI guidelines changed previous recommendations for patients with suspected ischemia and new or presumably new LBBB should not be treated as a STEMI equivalent

The purpose of our study was to determine the prevalence of AMI in patients who present with chest pain or an ischemic equivalent and have a new or presumed new LBBB.

NORMAL CARDIAC CELLULAR AND ELECTRICAL ACTIVITY

The excitability of the cells is due to the basic and well known cellular action potential.All normal cariac cells have a resting diastolic transmembrane potential of approximately -60 mv [SA nodal cells] to -90 mv [contractile cells]that is established by sodium potassium ATPase pump during phase 4 of the action potential.

This pump generates an electrolyte gradient with high potassium inside the cells and high sodium concentration outside of the cells.The charge on the membrane depends upon which of the two ions can freely cross the membrane.In diastole the cell is more permeable to K+ which provides for the nagative member potential.

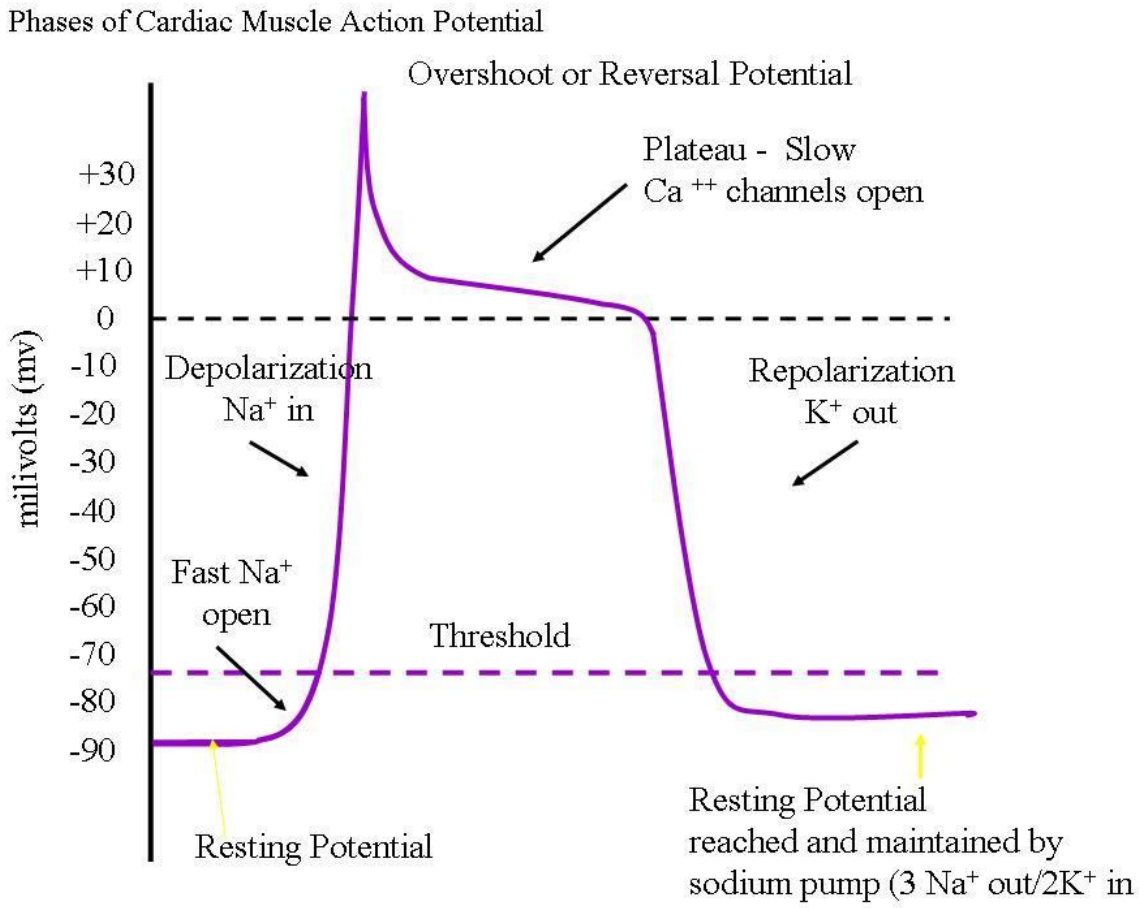


Fig 1 : Electric Potential of the cell

Automacity is generated in the non contractile pacemaker cells of the heart because of slowly depolarizing NA+ leak into cell during phase 4.The stable phase 4 diastolic transmembrane potential in contractile cells explains their lack of automacity and is what allows for their stable diastolic polarization.This is what produces the cellular polarity that is the foundation for the electrical vectors that ultimately forms the familiar QRS-T pattern on the ECG.When the membrane potential crosses a hreshold whether it is due to phase 4 leak or direct stimulus fast Na+ channels open and phase 0 depolarisationoccurs.The

transmembrane potential overshoots initially in phase 1 depolarisation reaching around +20 mv and then plateaus at 0mv during phase 2.This plateau is achieved by slow calcium influx balancing the slow K+ efflux and allows for prolonged contraction in cardiac myocytes.During phase 3 repolarisation calcium channels close and K+ efflux restores the negative resting membrane potential

After the initial stimulus on the cell the membrane doesn't completely and instantaneously depolarize through out the length of the cell. Rather it is a process that begins at the point of origin and continues three dimensionally towards the other pole of the cell.At rest the K+ permeability gives the cell membrane a negative potential which is analogous to having a positive charge on the outside of the cell.The cell membrane then becomes negative after depolarization.As depolarization progresses a dipole is setup with the vector oriented from the negative to positive portion of the cell.

A single positive electrode on the resting side of the cell would show a positive electrode deflection which would be greatest when the cell is halfway depolarized.In normal cells subsequent repolarization starts at the same location where depolarization began and progreses from the newly repolarized positive side to the still depolarized portion that is negative.

On a cellular level in contrast to clinical ECG the direction of the repolarization vector is oriented exactly opposite to depolarization vector.Myocardial cells contract when depolarized and their depolarization depolarizes succeeding cells leading to conductivity.

On an organ level the depolarization of the heart is represented electrocardiographically by the QRS and has a similar upright pattern as compared with cellular depolarization. Cardiac repolarization however is the opposite of cellular depolarization with a vector [T wave] that is normally positive.This is due to repolarization delay in the subendocardium resulting in a repolarizing wave moving away from the positive electrode and progressing from the subepicardium to the subendocardium.This delay is hypothesized to be due to a chronic hypometabolic state in the subendocardium secondary to poor blood supply which behaves electrically like ischemic tissue.The result is familiar upright QRS T pattern of the ECG.

ANATOMIC CORELATES OF ECG

Standard lead placement is essential for accurate measurement of ECG.

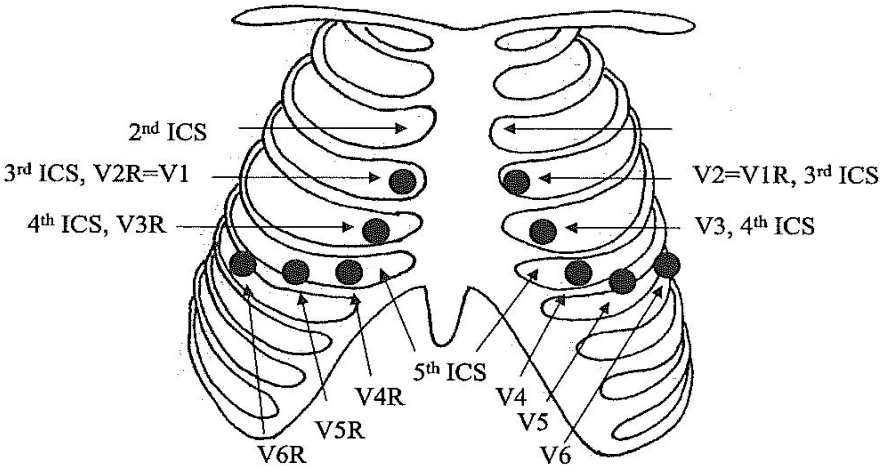


Fig 2 : Lead Placement on the Chest

One can relate the area of myocardium involved and coronary artery circulation affected with ECG changes.

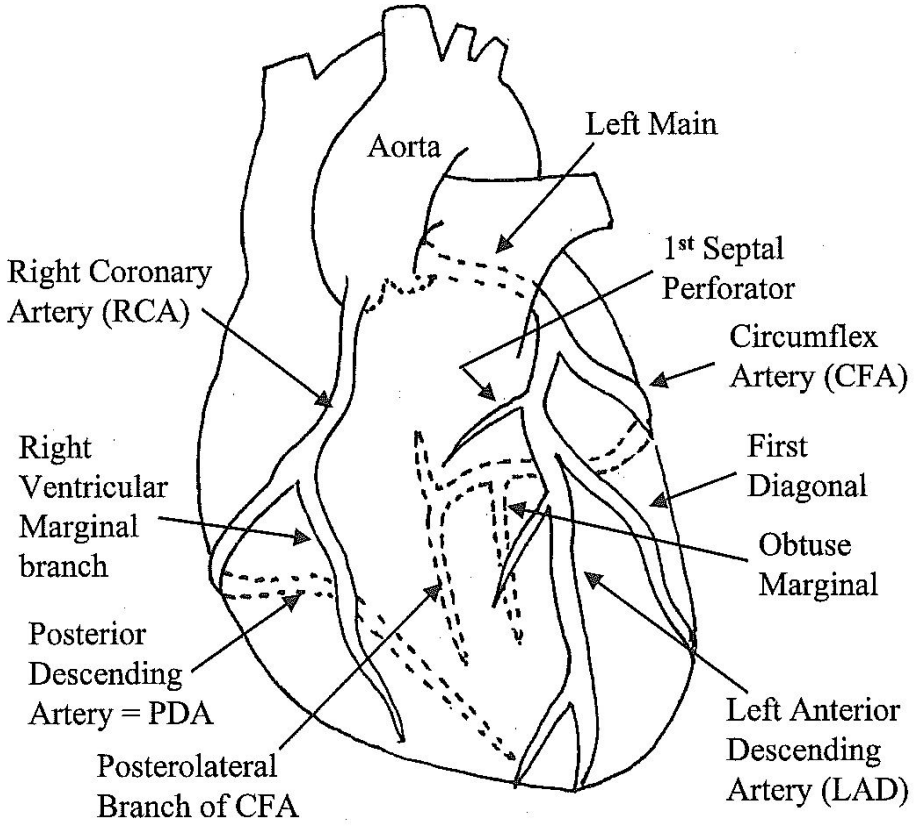


Fig 3 : coronary Anatomy

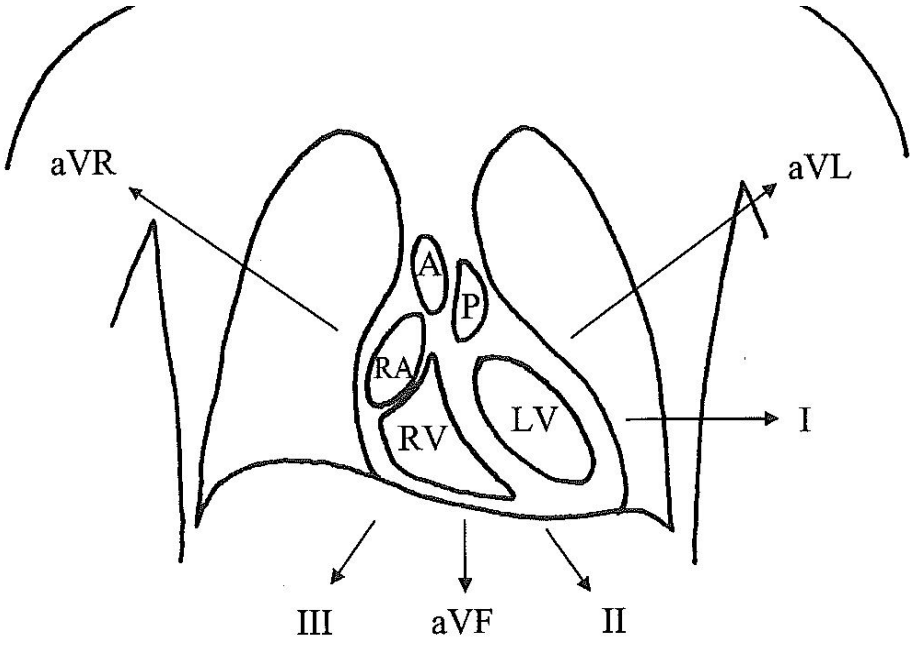


Fig 4 : Leads on the Heart

1. Inferior wall MI is identified by changes in lead III. Inferior wall MI often shows a ST elevation in lead III and ST depression in lead aVL unless there is concurrent lateral wall injury.
2. Lead aVL is best for detecting high lateral wall MI. Lateral wall MI is also detected in leads I and V5 V6
3. Septal MI is detected by changes in lead V1
4. Anterior wll MI causes ST elevation in V2 – V4
5. RV AMI causes ST elevation in V1. Right sided ECG shows reversal of V1 and V2.
6. Posterior wall AMI manifests as ST depression in V1 – V3

TABLE 1. ST ELEVATION, LOCATION OF STEMI, AND CORRESPONDING CORONARY ARTERY

ST elevation	Coronary artery (See Fig. 4-5)	AMI location
II, III, aVF (reciprocal ST depression in aVL)	RCA or circumflex artery	Inferior AMI
II, III, aVF (reciprocal ST depression in aVL) plus V1 Right-sided ECG: V4R	RCA proximal to RV marginal branch	Inferior and RV AMI
II, III, aVF (reciprocal ST depression in aVL) plus ST depression in V1–V4	Dominant RCA (70%) Dominant circumflex (30%)	Inferoposterior AMI
II, III, aVF plus (I, aVL and/or V5, V6)	Dominant circumflex **or** dominant RCA with lateral branches	Inferolateral AMI
II, III, aVF plus (V5, V6) and/or (I, aVL) and ST depression any of V1–V6	Dominant RCA with lateral branches or dominant circumflex	Inferoposterolateral AMI
V2–V4	Mid-LAD	Anterior AMI
I, aVL, V5, and/or V6	First diagonal or circumflex or obtuse marginal artery	Lateral AMI
V1–V4	LAD distal to first septal perforator but proximal to first diagonal	Anteroseptal AMI
V1–V6, I, aVL	LAD proximal to first diagonal and first septal perforator	Anteroseptal-lateral AMI

[a]There is great variation among patients. Use this for guidelines only

Ischemic processes

A cascade of events occurs in ischemic cells beginning with metabolic changes and subsequent electrical changes and alterations in contractility. Intracellular K+ decreases because of the decreased activity of the NA+/K+ ATPase pump. This causes a decrease in diastolic transmembrane potential and results in delayed phase 3 repolarization. Additionally in ischemic cells repolarization originates at the last point in the cell membrane to depolarize unlike a normal cell in which repolarization begins at the same point that depolarization began. Therefore in ischemia the cellular repolarization vector will be opposite to that of a normal cell and will be delayed.

Hyperacute T waves and T waves inversion

Subendocardial ischemia is the first pathologic process to have an ECG correlate during acute coronary syndrome. Endocardial ischemia results in delayed recoverey of this area.Since repolarization normally proceeds from the epicardium to the endocardium the direction of the repolarization vector does not change but the amplitude of the vector increases resulting in a hyperacute T wave.Subepicardial ischemia results in delay subepicardial repolarization and a reverse in direction of the repolarization vector;the result in this situation is T wave inversion.Subepicardial ischemia rarely occurs without involvement of

the endocardium[transmural ischemia].However the ECG picture is dominated by subepicardial ischemia.This is due most likely to the closer proximity of the epicardium to the electrode as well as to the relative mass of the subepicardium as compared with the endocardium.On the standard 12 lead ECG this pattern holds true for inferior ,lateral and anteroor ischemia whereas the reverse is true for posterior wall ischemia.In the anterior leads [V1 - V2] posterior subepicardial ischemia causes a positive T wave, whereas subendocardial ischemia results in a more negative T wave

ST segment Changes Two theories

The current of injury theory explains the ST segment changes during myocardial injury.Damaged cells lose their membrane integrity resilting in K+ leak. The outside of cell becomes more electrically negative which decreases the charge of the baseline or isoelectric line. If the damage is near positive electrode as in subepicardial/transmural injury the baseline TP segment decrease in voltage and the ST segment is then elevated relative to this base line. Since the cells still depolarize the ST segment remains at normal isoelectric line .In subendocardial injury the tissue becomes more negative at a distance from the electrode causing electrode resulting in a more positive vector for the isoelectric

line .In this situation the isoelectric line increases and the ST segment appears depressed.

The incomplete depolarization theory is based on the belief that damaged cells donot completely depolarize.If the electrode is in close proximity to damaged cells as in subepicardial/transmural injury a more positive ST segment will be maintained during systole.The partial depolarization of subendocardial injury would result in ST depression in leads over the normally depolarizing epicardium.With this theory there is no change to iso electric line.

Reciprocal changes

When the transmural injury occurs in addition to ST segment elevations at proximate electrodes, distant electrodes may show ST depression .These reciprocal changes of transmural injury can be explained by the preceding theories.There must be ST elevation at some recording electrode for ST depressions to be called reciprocal changes rather than simply subendocardial injury.

Q Waves

QRS changes in the first 40 ms of depolarization can be due either to preexcitation or infarction.Usually the degree of infarction necessary to result in pathologic Q waves is only seen in transmural

infarctions.Normal ventricular depolarization begins in the septum for the initial 20 ms.proceed to anterior inferior and lateral walls over the next 30 to 40 ms and terminates in posterior and high lateral walls.In the case of infarction the dead tissue becomes electrically silent.The vector for the initial 40 ms points away from the quiet tissue towards normal muscle resulting in classic ECG changes.In inferior and lateral infarcts the result is ep pathologic Q waves.Anterior septal infarcts will lose thweir R wave progression across the precordium and the loss of initial septal Q waves in Leads I,Avl,v5,v6.Anterior wall infarcts results in forces deviated to the right with an R wave in v1,while maintaining normal septal Q waves in leads I,Avl,v5,v6.

ANTERIOR WALL MI

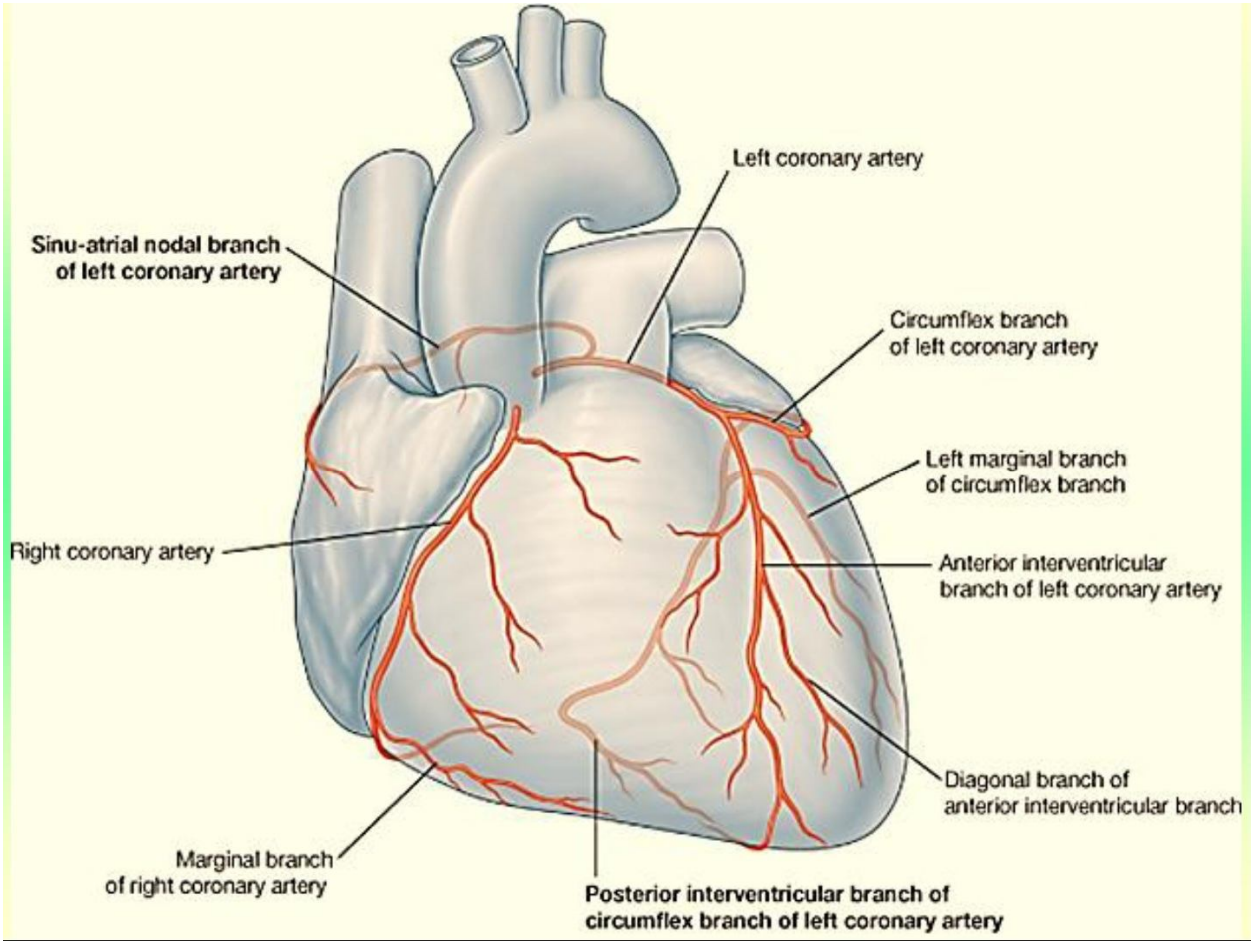


Fig 4 : Blood supply of the anterior wall of the heart.

Left main coronary artery supplies left anterior descending and circumflex arteries.

Proximal LAD occlusion causes lateral AMI , anteroseptal AMI or antero septal lateral AMI while mid LAD occlusion causes AMI.

ECG CHANGES IN ANTERIOR AMI

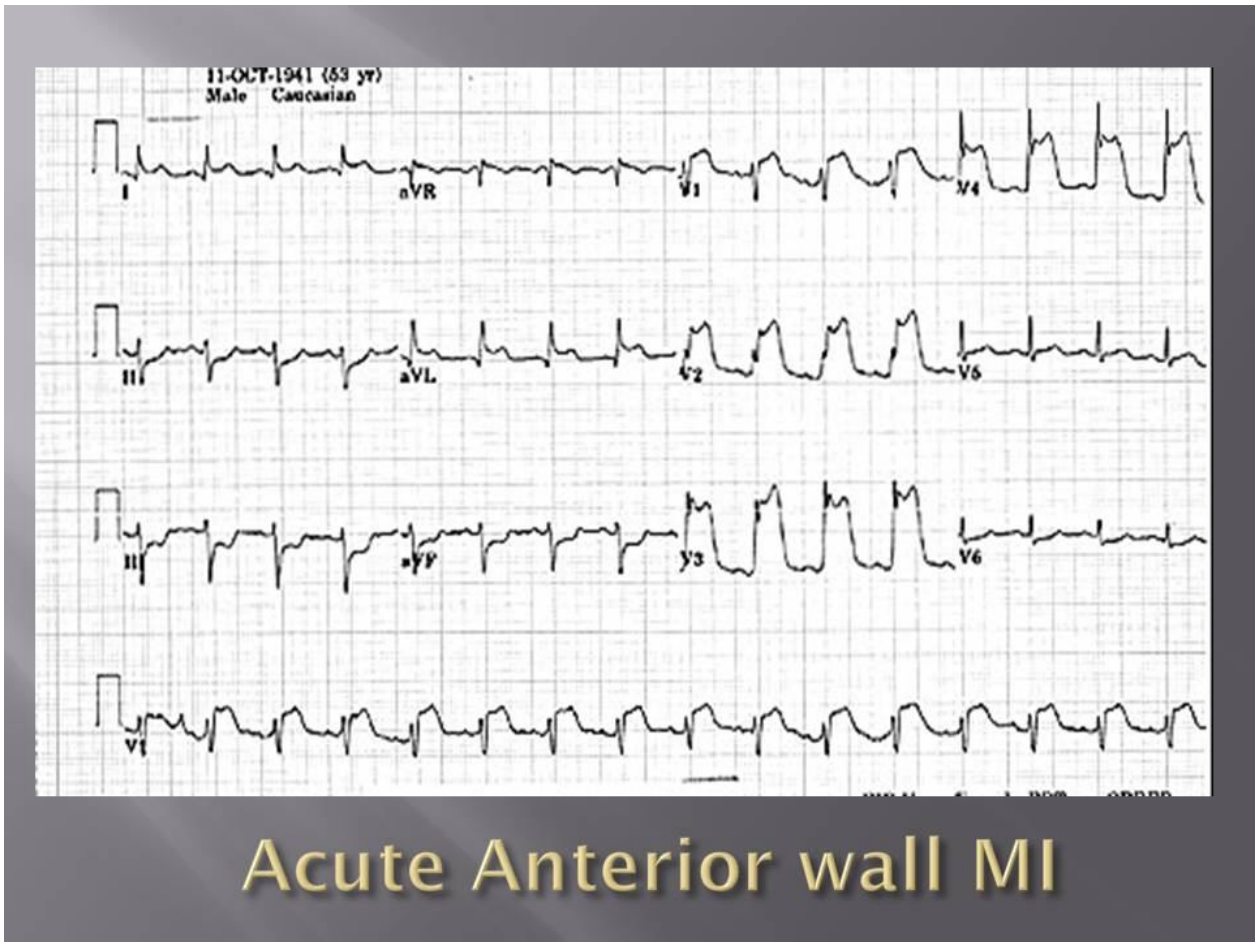


Fig 5 : ECG CHANGES IN ANTERIOR AMI

ST ELEVATION changes are seen in

1. Anterior AMI -V2- V4
2. Anterolateral AMI- changes are noted in V1- V4, V5 V6 and/or I and aVL
3. Anteroseptal AMI- elevation noted in V1- V4. Whenever ST elevation is seen in aVR it is indicative of occlusion proximal to 1st septal perforator.

4. Anteroinferior wall AMI manifests as ST elevation in V2- V4 and in II III and aVF.

PSUEDO ANTEROSEPTAL AMI

Pseudo anteroseptal AMI is one that looks like anterior AMI but is actually large high risk right ventricular AMI due to occlusion of proximal right coronary artery.

It manifests as

1. ST elevation in V1 – V3 (+_V4 V5)
2. Development of Q waves in V1-V3
3. Occurs with concomitant inferior AMI usually recognised by ST elevations in II III and aVF.

INFERIOR ST DEPRESSION CHANGES NOTED IN ANTERIOR MI

1. ST depression > 1mm in lead II is predictive of occlusion proximal to the first diagonal artery
2. ST depression in lead III ST elevation in aVL
3. Presence of reciprocal changes is associated with higher precordial elevation and often carries a poor prognosis.

ANTERIOR WALL AMI LOOK ALIKES

It often mimics early repolarisation , LVH, ventricular aneurysms and occasionally pericaditis.

PREDICTORS OF MORTALITY IN AMI

Overall mortality of patients treated with thrombolytics is 9.9%. the predictors of greater mortality are

1. Greater number of leads with ST elevation
2. Greater height of ST segments
3. Presence of reciprocal ST depression
4. Sum of absolute ST segment variations greater than 1.2 mv
5. Terminal QRS distortion by ST segment.

These factors are also indicative of increased benefits of reperfusion therapy.

LEFT BUNDLE – BRANCH BLOCK

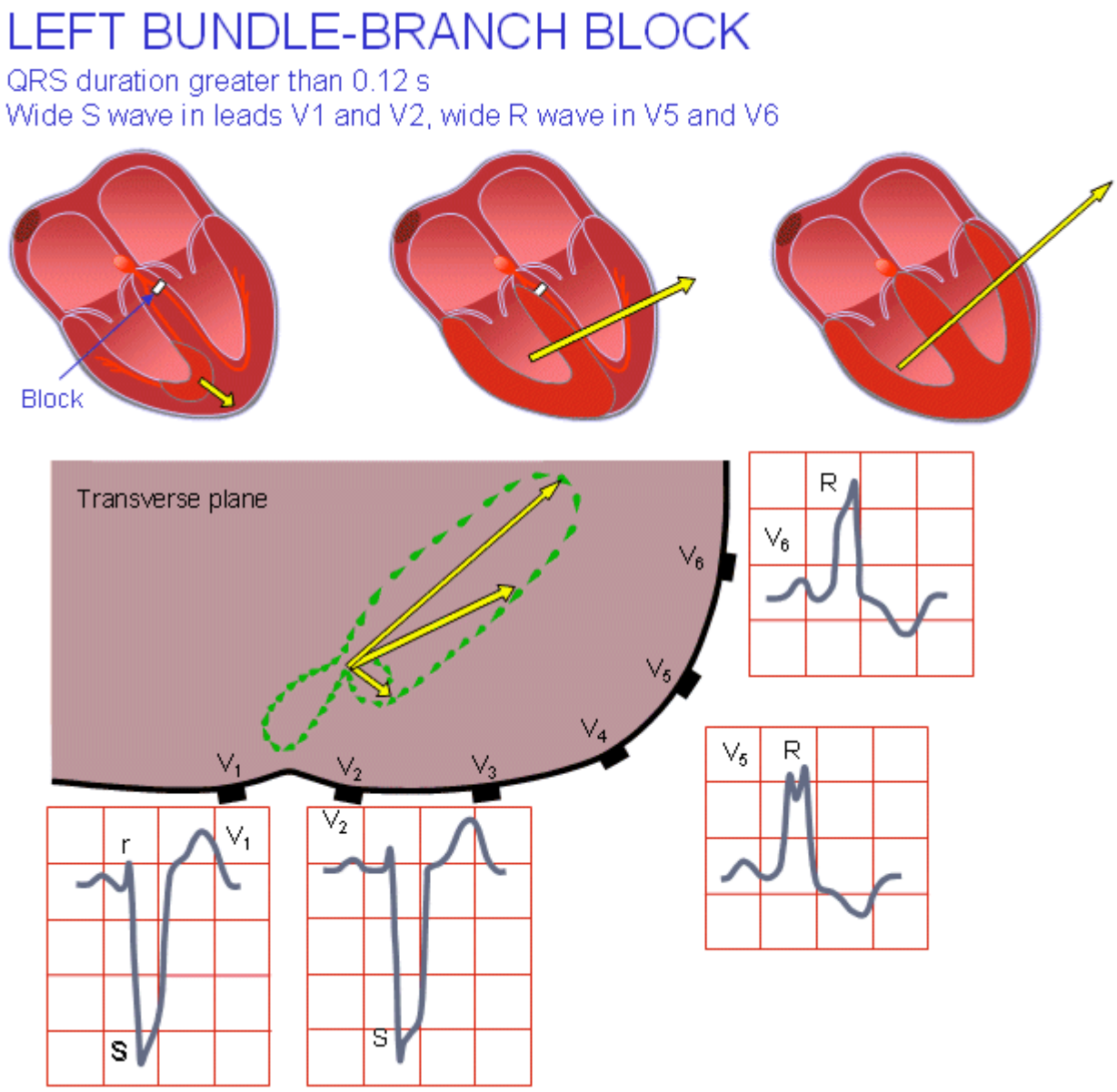


Fig 6 : Left Bundle – Branch Block

LBBB is a conduction abnormality in which specialized conducting fibers of the left bundle are non functional resulting in electrical propogation through slow conducting myocardium.

LBBB may be due to many disease states including cardiomyopathy and AMI.Unlike RBBB, LBBB with or without AMI

typically manifests ST elevation in the absence of any ischemia or infarction.Consequently the specificity of ST elevation for AMI when there is concurrent LBBB is much less than when there is normal conduction.

LBBB

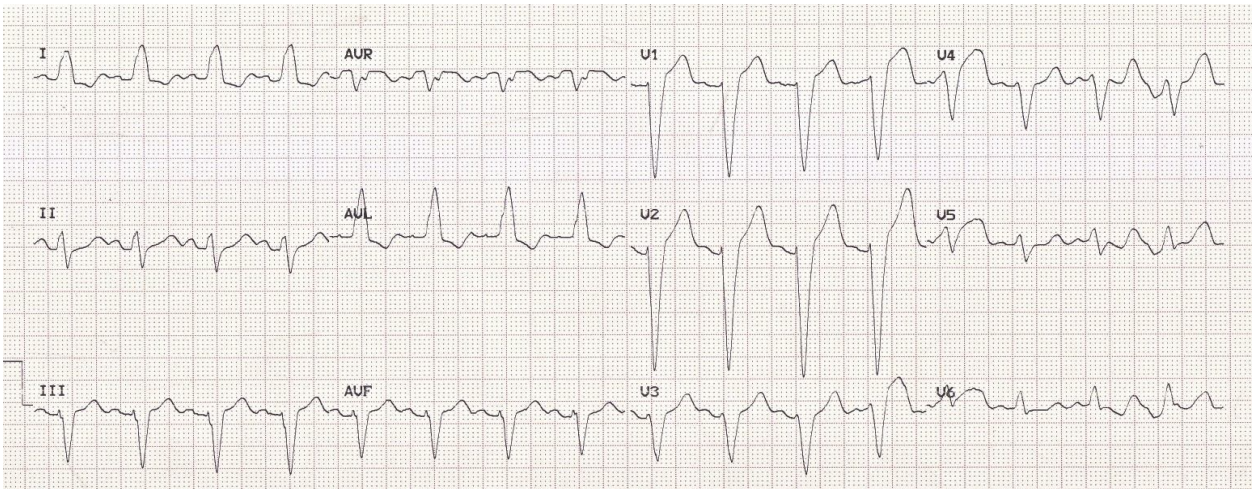


Fig 7 : ECG of Left Bundle – Branch Block

ECG diagnosis of LBBB

In LBBB with or without AMI the leftward component of the ECG complex is wide due to the propogation of depolarization through slow conducting myocardium.

Criteria for Diagnosis of LBBB

The following are criteria that must be present for the diagnosis of LBBBB.

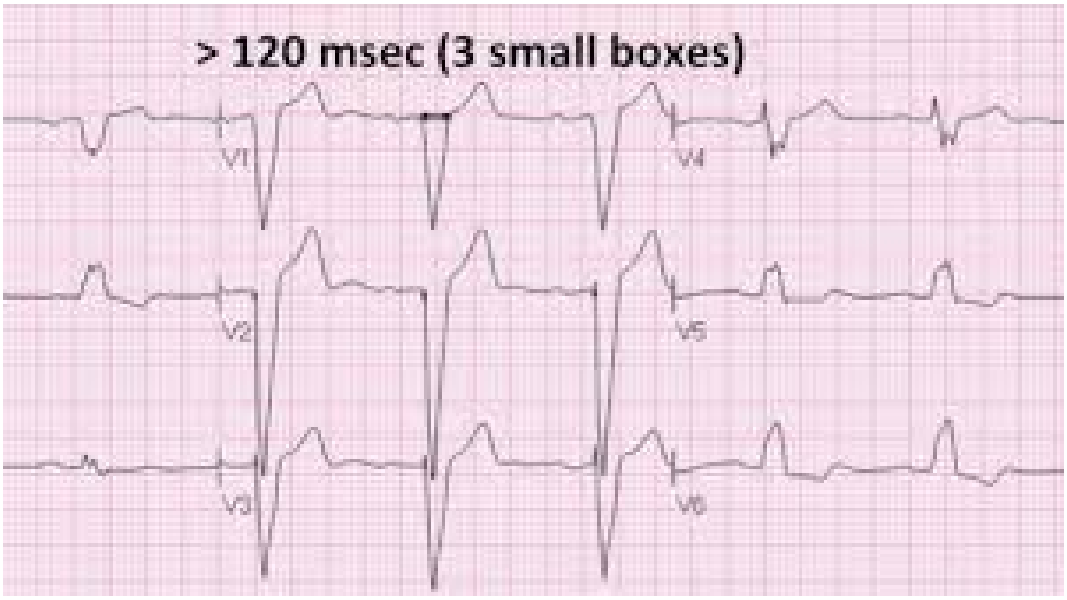


Fig 8 : Wide QRS complex

QRS > 120 ms

Lead I wide monophasic R wave

Any Q wave in Leads I,avl,V5 or V6 due to presence of MI,not result of old LBBB

Delayed intrinsicoid deflection in leads

V5-V6[>O.O4 sec to peak of R]

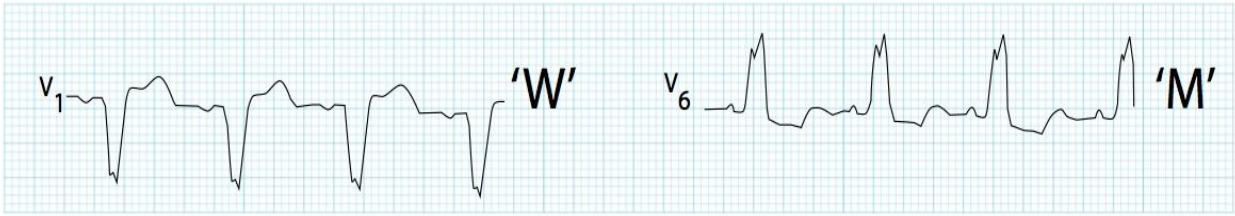


Fig 8 : W Pattern & M Pattern

The following features are typicaly present

V1; QS or RS pattern with large ,deep s wave

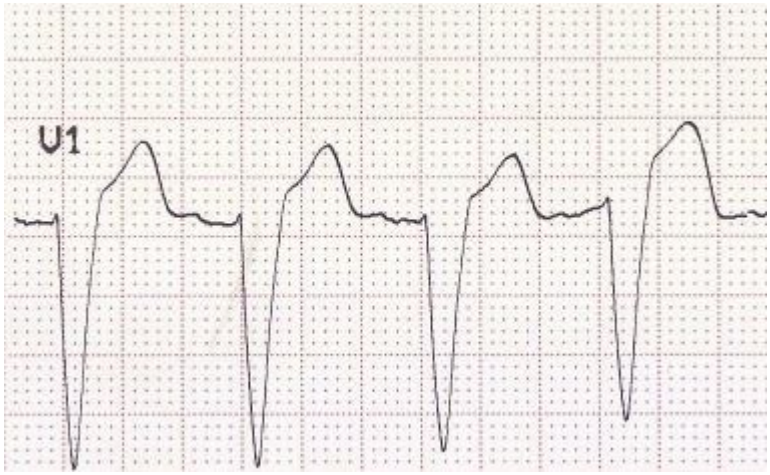


Fig 9 : Changes in V1

Left axis deviation

Discordant ST deviation and T wave inversion. Uncomplicated LBBB almost always manifest ST deviation and discordant T waves which means that ST segment and T waves are in direction opposite to the predominance of QRS complex.The magnitude of discordance should be proportional to the voltage of the QR.When discordance is not present ischemia or infarction should be suspected

LBBB Variations

Incomplete LBBB manifest a QRS duration of 100 to 120 ms other criteria are identical to those listed for LBBB

Rate limited LBBB may occur if the heart rate is too fast usually >100 beats per minute to allow repolarization of an abnormal conduction system with a prolonged refractory period .In these cases BBB may resolve with slowing of heart rate ,thus making ECG diagnosis easier

Intraventricular conduction delay is a term used to describe a wide QRS complex [100 ms or more] that occurs with no specific morphology

LBBB WITH AMI

Clinical factors

The prevalence of AMI in patients with ischemic symptoms and LBBB depends on the clinical situation.

LBBB is associated with chronic ischemic and non ischemic cardiomyopathy as wellas with AMI.A history of congestive cardiac failure ,dilated cardiomyopathy ,low ejection fraction or a chest film revealing cardiomegaly provides evidence that the LBBB is not new .Because LBBB may be present in many patients with nonischemic CHF,if the LBBB in not known to be new and there are no specific ECG findings of AMI ,it is important to ensure that the patient is

experiencing typical and ongoing symptoms before administering thrombolytics.

LBBB occurs in 6.7% of all AMI as diagnosed by CK MB.LBBB detected during continuous monitoring for 36 to 72 hours after thrombolytics may appear atleast transiently in upto 10.5% of AMI patients.Compared with patients with AMI and neither RBBB or LBBB patients with AMI and LBBB are older , more likely to present without chest pain.As diagnosed by CKMB only 8.4% to 16.6% of patients with LBBB and AMI receive any reperfusion therapy,most commonly because of non diagnostic ECG.This is in contrast to 32% of patients with AMI and no BBB who receive reperfusion therapy.Patients with LBBB and AMI are most likely to receive aspirin and beta blockers than patients with AMI and no BBB .LBBB has been to shown to be associated with LAD occlusion upto 43% of patients with LBBB and AMI.

Many physicians donot even attempt ECG diagnosis of AMI with LBBB because it is frequently taught that it is not possible.The notorious reputation of LBBB Ffor obscuring the diagnosis of AMI is due in large part to the difficulty of diagnosing previous MI in the presence of LBBB because LBBB alters Q wave patterns.Acute MI is also difficult to diagnose in the presence of LBBB because LBBB

typically manifests ST segment deviation.ST elevation due to the presence of AMI may be misinterpreted as due to LBBB and conversely the ST elevation of LBBB may be interpreted as due to AMI.

Specific criteria for AMI with LBBB

Specific criteria depend upon concordance and discornce .Consordance refers to the ST and or T wave being in the same direction as the majority of the QRS.ST and T wave discordance is the normal condition of LBBB.Although the presence of concordant T waves is abnormal and may be seen in AMI it is not very specific.The following morphologies have good specificity for LBBB with AMI and although the sensitivity is debated they have performed nearly as well in some studies as ST elevation for STEMI without LBBB

Concordant ST elevation >1 mm in one or more leads which means ST elevation in leads in which QRS is predominatly positive [v5,v6,I,Avl,II].This is diagnostic of AMI and contrasts with LBBB with no AMI which manifests discordant ST deviation

Concordadnt ST depression >1mm in one or more leads which means ST depression

In leads in which QRS is predominatly negative [V1,V2,V3,V4].This is 90% specific for AMI due to posterior injury

Discordant ST elevation >5 mm and disproportionate with QRS voltage appears to be 85% to 90% specific for AMI.This contrasts with uncomplicated LBBB,in which discordant ST elevation is typically <5 mm.

Diagnostic changes from a previous ECG

AMI superimposed on chronic LBBB may show diagnostic changes.If a prior ECG showed LBBB without ST segment deviation ,new ST elevation is diagnostic of AMI.If a previous ECG shows LBBB with discordant ST segment deviation,increased ST elevation may be as sensitive for AMI as increased ST elevation in patients without LBBB.If no previous ECG is available for comparision record serial ECGS or perform continuous ST segment monitoring .Changwes typical of AMI on serial ECG or continuous monitoring may be diagnostic

Essential considerations in the management of suspected AMI with LBBB

- Clinical suspicion of AMI
- Specific Criterias [sgarbossa,Smith]for AMI
- Availability of angioplasty
- Thrombolytic Contraindications

Clinical Significance

LBBB is usually secondary to underlying heart disease. Approximately 30% of patients due to heart failure .

70% had preceding evidence of LVH. 12% have no demonstrable heart disease.LBBB has higher mortality from heart failure and infarction even in persons with no overt heart disease.As the duration of QRS complex widens the risk of heart failure increases.Thre is a higher risk of high grade atrioventicular block and cardiac death nad one of the important cause of sudden cardiac death.Among patients with coronary artery disease LBBB signifies more exterior wall damage more severe LVD and increased mortality and morbidity

Patient with axis deviation have severe clinical manifestation.Left axis deviation is associated with severe conduction system because it includes fascicles and main left bundle .Right axis deviation suggests dilated cardiomyopathy with biventricular enlargement

The abnormal ventricular activation incites compromisd hemodynamic changes superimposing on the already diseased heart. There is premature movement of septum into left ventricle followed by delayed contraction of lateral and posterior left ventricular wall. As a

consequence when the lateral wall contracts blood pushes the compliant septum towards right ventricular cavity than the outflow tract reducing the ejection fraction and efficiency. The dysynergy in left ventricle is severe with a delay of more than 60 msec between septum and lateral wall contraction is found in 60% of patients with QRS durations of 120 to 150 msec and in 70% of those with QRS more than 150 msec

A major impact of LBBB it obscures or simulates other ECG patterns . In LVH there is an increase in amplitude of QRS duration and there is left axis deviation intrinsic to LBBB.In myocardial infarction the emergence of abnormal Q waves with infarction is dependent on normal initial sequence of ventricular activation .In LBBB there is 1]absence of Q waves 2]low r wave amplitude in mid precordial leads3]ST T wave changes

All these can simulate anterior infarction pattern

CARDIAC BIOMARKERS

The diagnosis of acute MI is mainly made upon ECG findings or clinical picture (angina) or cardiac biomarkers out of which the latter is more reliable as ECG can be non specific and clinical findings may not manifest. Therefore, the latest diagnosis of MI is made by elevated levels of cardiac biomarkers mainly CKMB and troponin T of which troponins are the preferred lot.

The diagnosis of MI using biomarkers depend upon a vast range of criteria

- The 99th percentile of the normal range of values should be the cutoff value for both troponin and CKMB
- A rise and/or fall should be observed and a value greater than 3 standard deviations is to be considered significant

A significant change is always pathological

Cardiac troponins rise nearly 2-3 hours after acute myocardial injury and an aggregated assay along with other markers like ck-mb and myogobin which are rapid markers released during muscle damage, though non specific, can help in diagnosis of acute MI.

Cardiac troponin I and T vary in sensitivity and specificity for biochemical damage.

Reversible and irreversible cardiac injuries are hypothesised to depend upon troponin and myoglobin values, and their relation is questioned. If biomarkers are produced the same way in both reactions the possibility in distinguishing them is reduced.

Troponn T and I are regulatory proteins that affect cardiac contractility by regulating calcium mediated channels and thereby affecting interactions between muscle proteins, actin and myosin and since they are products of a specific gene they are found to be unique for the myocardium out of which troponin I is even more specific as it is not found anhywhere except for cardiac tissue when compared to troponin T which may be found in certain groups of skeletal muscles too which accounts for slight elevation of troponin T during specific muscular damages which focusses the importance of assay of troponin I for diagnostic purposres. The store for troponin can be the cytosolic pool which accounts for its earlier release along with myoglobin and also structural pool that accounts for its sustained release.

Any variations in sensitivity and specificity of the assay can be accountable to the variations in standardisation or to the presence of modified troponin T and I present in the serum pool or to the variation of cross reacting antibodies which leads to development of high sensitive or very highly sensitive assays which when not specified

shows the assay to be contemporary assays which calls for standardisation of an assay which makes the assays imprecise due to the very low values of cardiac markers otherwise found in the subjects (eg, 0.01 to 0.5 mcg/l).

Young and healty individuals are found to have a small level of troponin elevated in ther blood which may be due to muscular activity which can again be questionable in an assay and defining normal range of troponin becomes even more difficult. Therefore an elevation above the 99^{th} percentile is considered to be an increased cardiovascular risk

Diabetes mellitus, left ventricular hypertrophy, chronic kidney disease, and heart failure are the major predictors of elevated troponin T values and the incidence of cardiovascular mortality is found to be more in such cases

Elevated levels of biomarkers can also be seen in supply demand abnormalities, hypertrophies of muscles and also in post operative and trauma patients which can give a false value in assays

Cardiac markers can be used for late diagnosis of infarctions or reinfarctions as tni can persist upto 10 days after an AMI favoring late diagnosis. Reinfarctions can be assessed by prompt re elevation of the troponin levels

An immediate measurement of cardiac troponin is mandatory in case of a reinfarction and a 20 % increase in second sample values confirms it.

Though troponin elevation is a marker for AMI it can have varied differential diagnosis since elevation fits into even moderate to severe pulmonary embolism with acute right heart overload, heart failure, and myocarditis.

In many studies the infart sizes has been measured by the level of troponins thereby signifying their importance in assessing the size of infarct.

Troponin T and I are considered to be good markers of prognosis of mi too and has equal range of depletion or change after an MI this was studied in the GUSTO 3 trials and repeat after 30 days showed subsequent regression in values of troponins studies have also showed that an increased rate of cardiac deaths are found in patients with elevated levels of troponins

In metaanalyses of deaths in patients with elevated troponin levels, a patient with elevated troponin T is found to be at higher risk of death when compared to the ones with an elevated troponin I value.

Persistent occlusion of the culprit vessel and a reduced left ventricular ejection fraction are associated with an increase in troponin value

Creatine kinase a biomarker that exists in M and B isomer forms and is a clinical guide in assessing the destruction of muscle tissues and is therefore not specific for MI. Though sensitivity and specificity are less for CKMB it is utilized because of easy and rapid assessment.

A negative analysis can turn up as CKS are elevated also in certain muscular dystrophies, adenomyositis and poliomyositis which can act as a differential diagnosis for ami in terms of elevated serum biomarkers.

They elevate usually at about 4- 6 hours after the cardiac injury but may not be elevated upto 12 hours in some patients. It returns to baseline in over 36 to 48 hours but they have to be excluded in cases with skeletal muscle injury as such injuries themselves can cause an elevation in the b fraction of CKMB and therefore in CK level.

The grade of reperfusion can also be assesed with the elevated levels of CK.

Resampling of CKMB can be used to assess reinfarction along with troponin as it is elevated in such cases

A crusade initiative in UK did studies on the same the findings are as follows

1. "Results in them were discordant in 28 percent of patients. The more sensitive was troponin, as 18 percent had normal CK-MB values but elevated troponin. In addition, 10 percent had false positive CK-MB elevations, as defined by normal troponin values."
2. "Compared to patients who were negative for both biomarkers, in-hospital mortality was not increased in patients who were troponin-negative and CKMB-positive (ie, false positives; 3.0 versus 2.7 percent, adjusted odds ratio 1.02, 95 percent CI 0.75-1.38)."
3. "Compared to patients who were negative for both biomarkers, there was a nonsignificant trend toward increased mortality in patients who were troponin-positive /ck-mb-negative (4.5 versus 2.7 percent, adjusted odds ratio 1.15, 95 percent CI 0.86-1.54) and a significant increase in mortality in patients who were positive for both 263+ Biomarkers (5.9 versus 2.7 percent, adjusted odds ratio 1.53, 95 percent CI 1.18-1.98)."

The studies finally conclude the superiority of troponin over CK-MB and the use of cardiac biomarkers in assesing the time of AMI, the size of infarct,the amount of reperfusion and the prognosis of MI.

ECHOCARDIOGRAPHY IN REPERFUSION DECISION

Inadequate coronary blood flow leads to ischemic myocardium and abnormal contraction or regional wall motion abnormalities which is detectable with echocardiography.A wall motion abnormality may manifest as hypokinesis [decreased wall motion]akinesis[no wall motion] or dyskinesis[bulging] or aneurysmal diastolic distortion.

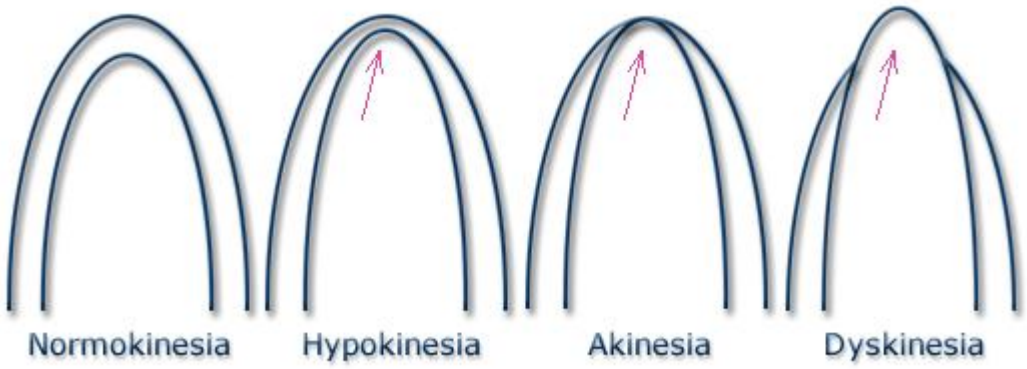


Fig 10 : Wall motion abnormalities on echo.

The presence of a WMA cannot distinguish AMI from old MI. However when the ECG differential diagnosis is AMI versus "old MI with persistent ST elevation",the presence of diastolic distortion with myocardial thinning and dyskinesis strongly supports a diagnosis of old MI

The presence of new regional WMA supports the diagnosis of myocardial ischemia and in conjunction with new ST elevation in the corresponding ECG location, strongly suggests STEMI. A new WMA in conjunction with equivocal ECG, in the right clinical setting is an indication for reperfusion. Normal wall motion rules out large risk area but echocardiography is insensitive for small risk areas, so normal wall motion in the presence of diagnostic ECG doesn't rules out a small STEMI. Nonethless in the presence of an equivocal ECG, normal wall motion on an echocardiogram of good quality detected by a skilled technician and interpreter woulD provide evidence against AMI and be a good reason to withhold thrombolytics.

Almost all the segments of the left ventricle can be visualized by by parasternal. apical and sometimes subcostal windows. For the purpose of regional wall motion analysis wall motion score index is calculated to quantify the magnitude of region wall motion abnormalities.

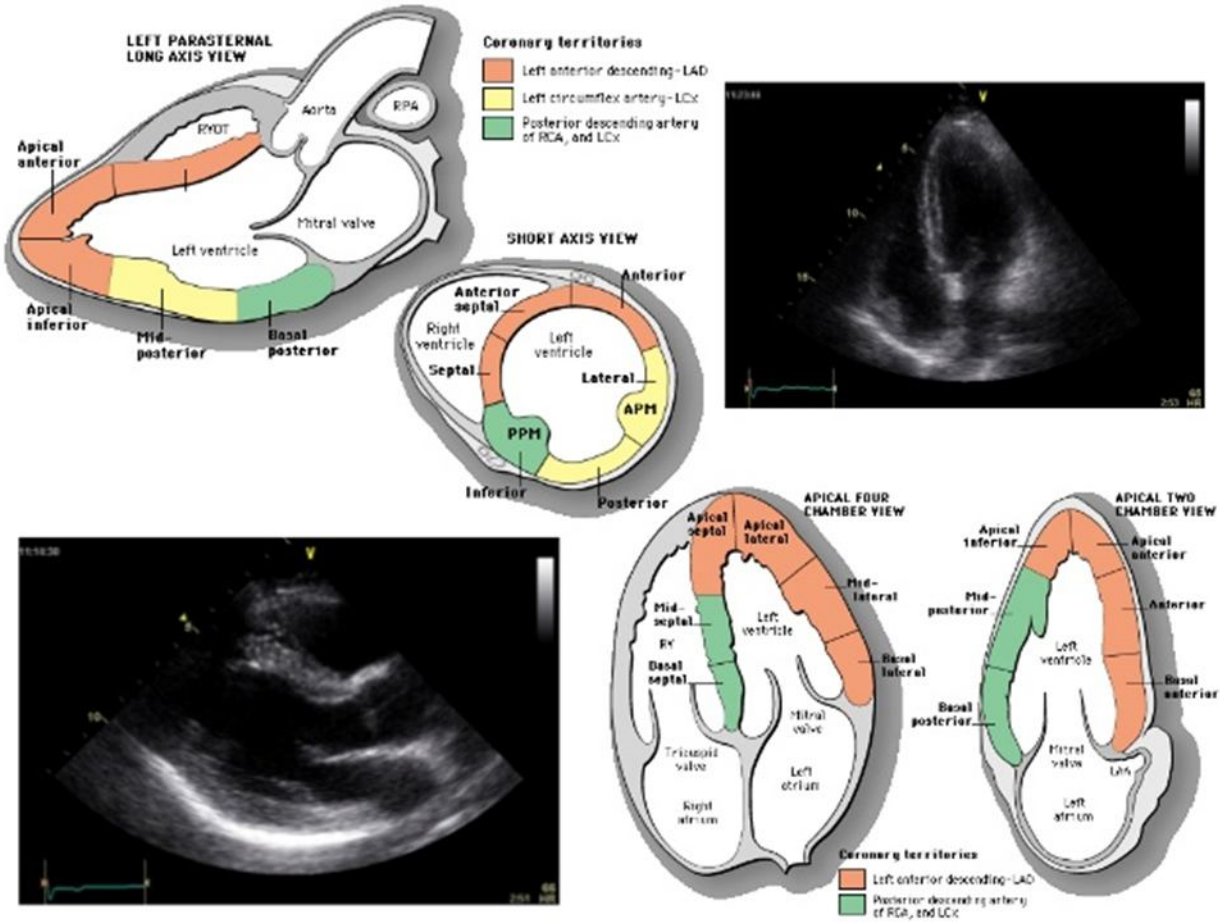


Fig 11 : Views of Heart

Echo in Acute Myocardial Infarction

In patients with acute coronary echocardiography has several purposes

1) For the diagnosis or excluding acute MI in patients with persistent angina and when the ECG is ambiguous.
2) Quantifying the magnitude of the myocardial segment at risk and the ultimate infarct size after reperfusion
3) Evaluating the complication of Infarct
4) Assesing the viability of the affected myocardium
5) Risk stratification

2D echocardiogram are helpful in following up the reperfused myocardial or expansion of the infarct size in patients with STEMI.Prolonged akinesis doesn't mean failure of reperfusion.In these cases low dose dobutamine contrast or strain imaging may be helpful in detecting viability.

ECHOCARDIOGRAPHY AND ECG

Echocardiography helps in evaluation of ischemic symptoms and in reperfusion decisions in following conditions.

1. Non specific ST elevation consistent with AMI
    a. If there is a new WMA patient is a candidate for reperfusion therapy.
    b. If wall motion is normal it makes AMI of large risk area unlikely
2. Precordial ST depression that is consistent with either posterior STEMI or anterior UA/ NSTEMI
    a. If there is a new posterior WMA the patient is a candidate for reperfusion therapy.
    b. If there is new anterior WMA it is indicative of an UA/ NSEMI not indicating thrombolytic therapy
3. When ECG is non specific for any kind of ACS then the patient is not eligible for thrombolytics but is indicative of angiography +_PCI

4. When ST elevation an QS waves are present and differential diagnosis is AMI versus ventricular aneurysms

    a. Presence of diastolic distortion and myocardial thickening supports ventricular aneurysm and ST elevation is probably old.

    b. Akinesis or hypokinesis without diskinesis however does not necessarily imply that ECG findings are new.

LIMITATIONS OF ECHOCARDIOGRAPHY

1. Echocardiography cannot differentiate between a STEMI and UA/ NSTEMI
2. Echocardiography cannot determine the time course of AMI.

HISTORICAL PERSPECTIVE

Recognising left bundle branch block in Acute myocardial infarction was made by Oppenheimer and Rothschild way back in 1917.Patients with LBBB presents an unique problem in that they are relatively older with other co morbidities like systemic hypertension, Diabetes Mellitus, Heart failure etc.Even though there is an observed increased mortality in these subgroups .question remains whether it is due to confounding factors like age and other comorbid conditions

Diagnosis of MI in the background of LBBB always remains challenging as activation of left ventricle happens much later and the initial wavefront activating the septum moves from right to left and as a consequence the Q waves the signature of AMI are absent. Also secondary ST T wave abnormalities obscures injury currents of ischemia and infarction

In 1996 sgarbossa came out with the electrocardiographic criteria for the diagnosis of Myocardial Infarction in patients with Lbbb and came out with the concept that ST deviation is the ony useful electrocardiographic finding that would be helpful in the diagnosis of Acute MI in LBBB.They reviewed 26,003 patients enrolled in GUSTO 1 [Global Utilization of Streptokinase and Tissue Plasminogen Activator of Occluded Coronary Arteries] and compared people who

had LBBB with Acute MI with electrocardiograms of control patients who had chronic coronary artery disease and left bundle branch block. Derivation set included 131 patients [0.5%] with LBBB.Average time from onset of symptoms to ECG was 120 minutes. Validation set was 45 patients from GUSTO 2A with AMI and LBBB.

Concept of concordance and discordance: It refers to whether the last portion of QRS complex goes in the same direction as that of T wave .Discordance is the rule which denotes secondary changes. Concordance is always due to primary ischemia/inf arction.

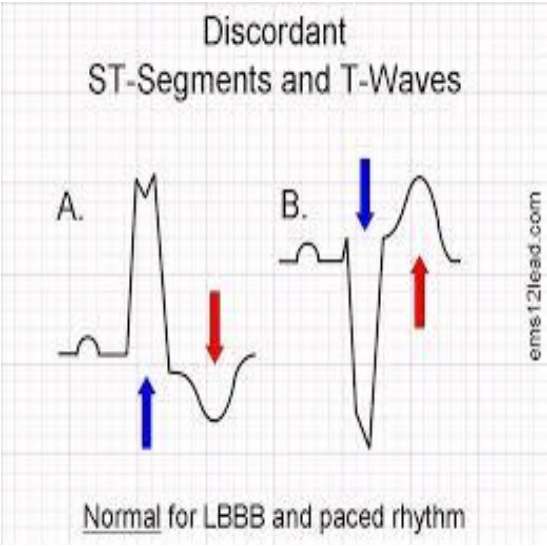


Fig 12 : Normal Discordance

They came out with a cut off point for ST segment deviation that would differentiate between LBBB patients with acute infarction and those without infarction in the absence of previous ECGS.

Identified three predictive criteria

1) “ST segment elevation greater than or equal to 1mm concordant with QRS
2) ST segment depression greater or equal to 1mm concordant with QRS
3) ST segment elevation greater than or equal to 5mm discordant with QRS”

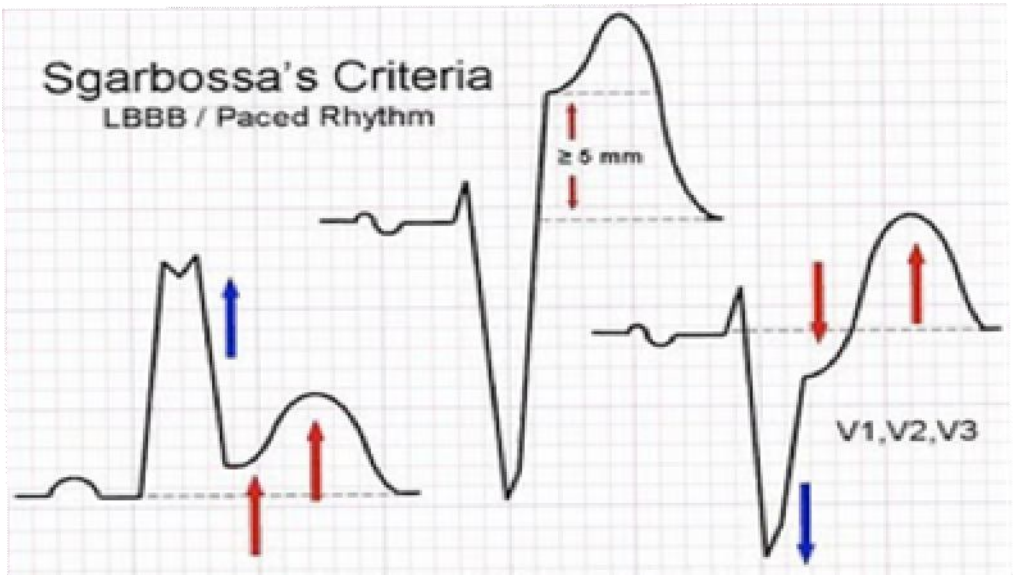


Fig 13 : Sgarbossa's Criteria

Of these the ST elevation concordant with major QRS deflection I any lead had odds ratio for AMI of 25.2 and ST depression in the the anterior leads v1 v2 or v3 had odds ratio for AMI of 6 .Discordant ST segment elevation of 5mm or more in any lea had a weaker association of odds ratio of 4.3.when combined together these three criteria had a sensitivity of 78% and specificity of 90%.

The presence of LBBB in patients with acute MI almost always associated with increased morbidity and mortality.If its new then it is due to occlusion of proximal LAD which means an extensive anterior wall MI with a large volume of myocardial involvement.If its an old LBBB it's a strong marker of compromised left ventricular dysfunction and any further loss of myocardial involvement will lead to hemodynamic jeopardy.

MODIFIED SMITHS CRITERIA

The low sensitivity in Sgarbossa criteria is due to

The study is based on the enzyme diagnosis of Infarction and not angiography so the infarction group included both ST elevation and Non ST elevation Infarction

1) Anterior MI is diagnosed by elevation of ST segments in v1 to v4. In LBBB there is a preexistent discordant ST elevation.The excessive ST elevation has to be assessed for concluding a diagnosis of most anterior Myocardial Infarction.Sgarboss used an absolute numerical value of 5mm for discordant ST elevation.
2) Smith proposed instead of using an absolute value use of a proportion criteria [any ST segment elevation to S ratio of less than 0.25 with atleast 1mm ST elevation improves the prediction of coronary occlusion.

3) 162 patients with LBBB chest pain were studied of which 33 had angiographic evidene of coronary occlusion. And 129 were control group.The new criteria has a positive likelihood of 9 and negative likelihood of 1.

Anterior Myocardial Infarction caused by occlusion of left anterior decending artery results in ST elevation in the leads v1 to v4 and may be in v5 , I,Avl if the occlusion is proximal to diagnol artery.In LBBB there is already a preexistence discordance with baseline ST elevation in V1 TO V4.In the setting of mid LAD occlusion the diagnosis of STEMI will be purely based on excessive discordance in v1 to v4.Sgarbossas weighted criteria gives only 2 points to this discordance rule and hence will miss a large number of infarctions..By replacing this absolute criteria with proportion criteria the sensitivity greatly improved

In this derivation study the ratio of ST segment elevation to S ratio proved to have significantly better sensitivity and accuracy than that of the maximum ST segment elevation.

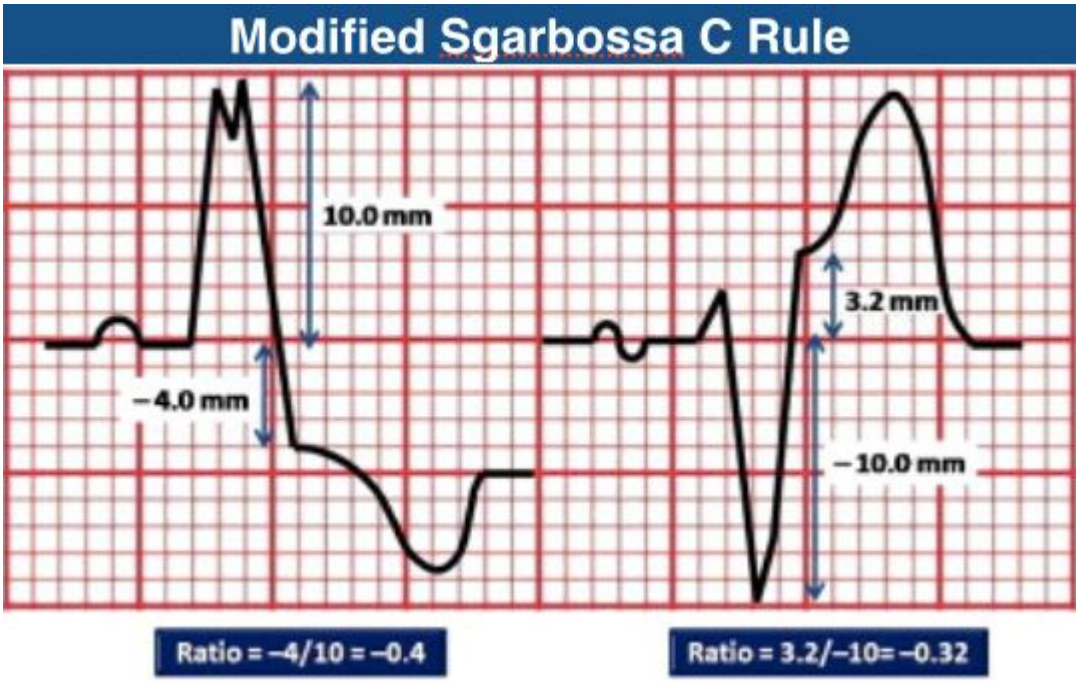


Fig 14: Modified sgarbossa

FTT collaborative group:. Indications of fibrinolytic therapy in suspected Acute MI ;collaborative overview of early mortality and major morbidity results from from all randomized trials of more than thousand patients 1994

Methods: FTT collaborative group reported the combined results of all 9 placebo controlled thrombolytic trials including ISIS 2 and GEISSI 1 .

Findings of 2146 cases of BBB,35day mortality for the thrombolytic group was 18.7 % v/s 23.6 % for placebo.The benefit associated with thrombolytic therapy for typical chest pain with LBBB far outweighs the associated risks

Comment - the benefit of thrombolytics for suspected and BBB was demonstrated almost 15 years ago , in the intervening years physicians have come to recognise atypical chest pain as a symptom of AMI and probably obtained many more ECGS than in the past .Though it has never been shown and would be impossible to show suspected AMI today may have a more liberal definition in the minds of physicians than 15 years ago .If so the pretest probability of AMI is lower and the recommendations for thrombolysis for every patients suspected AMI and LBBB would be too liberal , perhaps this accounts for the low prevalence of AMI with LBBB in Kontos et al, Li et al and Ozment et al .a more appropriate indication for reperfusion therapy is for those patients with symptoms suspicious for persistant coronary occlusion who have LBBB not known to be old.

Studies suggesting the utility of specific ECG criterion for AMI in LBBB IS low:

Shlipa MG et al. should the electrocardiogram be used to gate therapy for patients with LBBB or suspected MI?

Methods Shlipak et al retrospectively applied the algorithm of sgarbossa et al to 83 patients with LBBB who presented with acute chest pain, acute pulamonary oedema or cardiac arrest. AMI was

diagnosed by CKMB of 7ng/ml or cardiac troponin cTnI of 1.5 ng/ml or more .

Comment - this biomarker elevation will be caused by cardiac arrest or pulmonary oedema without coronary occlusion.

Findings - Sgarbossa criteria was insensitive for myocardial damage so defined . The results of this study was extrapolated to indicate every 1000 patients with LBBB and clinical presentation suggetive of AMI ,926 would survive without major stroke if all patients recieved thrombolysis . This is incontrast to the stroke free survival of only 918 patients if the scarbossa criteria was used to determine therapy.

Comment - The calculation was based on over high excess high stroke rate of 8.4v% per thousand. The actual overall excess non fatal stroke rate for tPA is closer to 3.5 per thousand if both infarct and haemorrhage are considered

Kontos the MC et al .Can Myocardial Infarction be rapidly identified in emergency department patients whi have left bundle ? 2001

Methods Kontos et al evaluated 7725 patients with ischemic symptoms over 6 years .They reviewed the incidence of LBBB ,LBBB and

AMI,and predictors of AMI in the presence of LBBB with special attention to new versus old LBBB and to Sgarbossa criteria.

Findings:LBBB was found in 182 patients.AMI was diagnosed in 24.Presence of one or more of the Sgarbossa criteria had sensitivity of 46% specificity of 93% PPV of 50% and NPV of 92%.New or indeterminate age LBBB had sensitivity of 83% specificity of 41%,PPV of 18% and NPV of 94%.Addition of positive initial CK MB to Sgarbossa criteria improved sensitivity to 63% with a specificity of 99% and PPV and NPV of 88% and 94%.Mean peak total CK and CKMB were 2750 IU/L and 212 ng/ml in the group with positive criteria and 370 IU/L and 32 ng/ml in the group without criteria though use of reperfusion therapy is not stated.

Comment: The authors considered the sensitivity of criteria unacceptably low identifying only 11 of 24 patients with AMI yet also having a poor PPV.They also were of the opinion that LBBB not known to be old performed poorly.

Table 2 : Prevalence of LBBB and AMI in studies of patients with suspected ACS

Study	Year	Total, no. of patients	No. of patients with LBBB (%)	No. of LBBB patients with AMI (%)
Sgarbossa et al. (1)	1996	26,003	145 (0.6)	131 (90)
Cannon et al. (2)	1997	1,416	127 (9)	40 (31)
Wong et al.[3)	2005	17,073	300 (1.8)	242 (81)
Al-Faleh et al. [4]	2006	22,839	267 (1.2)	158 (63)
Lopes et al. [5)	2011	5,742	98 (1.7)	85 (87)
Jain et al. [6]	2011	892	36 (4)	12 (33)
Subtotal (n=6)		**73,965**	**973 (1.3)**	**668 (67)**

Based on the previous studies the incidence of LBBB in those seeking medical attention for suspected acute coronary syndrome was 2%.Comparring to those patients without bundle branch block these patients tend to be older ,with associated comorbid conditions like diabetes,hypertension etc[18,20].Also patients with LBBB tend to have poor prognosis with adverse consequences like recurrent MI,stroke,death compared to those without LBBB.But it should be kept

in mind these cannot be contributed to LBBB itself rather it is the underlying preexisting pathology that caused the LBBB[structural,ischemic heart diseases]must be held responsible for the worse outcome.Since LBBB is more prevalent among the elderlies which in turn associated with other co morbidities LBBB can be considered one contributing integrative co factor than a independent cause for mortality or morbidity. Compared to left bundle branch block, Right Bundle Branch Block had higher mortality rate and the independent mortality risk of LBBB yet to be proved.[1,21]

Table 3 : Representative studies of patients with suspected AMI presenting to emergency department

Study	Year	Total, no. of patients	No. of patients with LBBB (%)	No. of LBBB patients with AMI (%)
Fesmire et al. (53)	1989	440	24 (5.5)	3 (13)
Otto and Aufderheide (54)	1994	428	18 (4.2)	5 (28)
Kudenchuk et al.(55)	1998	3,027	57 (1.9)	20 (35)
Edhouse et al. (56)	1999	797	50 (6)	26 (52)
Shlipak et al. (39)	1999	n/a	83 (100)	26 (31)
Li et al. (15)	2000	n/a	190 (100)	25 (13)
Kontos et al. (14)	2001	7,725	182 (2.4)	24 (13)
Gunnarsson et al. (43)	2001	n/a	158 (100)	76 (48)
Maynard et al. (57)	2003	n/a	56 (100)	18 (32)
Chang et al. (31)	2009	7,937	191 (2.4)	11 (6)
Bansilal et al. (1)	2011	2,271	102 (4.5)	5 (5)
Kontos et al. (34)	2011	n/a	401 (100)	116 (29)
Subtotal (n=13)		**26,322**	**802 (3.0)** *	**437 (26)** †
Total (n=19)		**100,287**	**1,775 (1.8)** *	**1,105 (42)** ‡

A substantial amount of patients of suspected acute coronary syndrome did not have a culprit artery occlusion on angiogram and should have been considered as non ACS cause/Unstable angina or NSTEMI. This has a significant clinical implication because these patients would have been unnecessarily subjected to thrombolysis or angiogram and and thrombolysis would have been proved harmful in case of NSTEMI.

Table 2 Clinical Outcomes in Patients With BBB in Randomized Controlled Trials of Intravenous Fibrinolytic Therapy Versus Standard Care in AMI*

	Death		Stroke		Bleeding	
Patient Population	Fibrinolytic	Control	Fibrinolytic	Control	Fibrinolytic	Control
All patients (n = 58,600)	2,820 (9.6)	3,357 (11.5)	340 (1.2)	224 (0.8)	325 (1.1)	111 (0.4)
Patients with BBB (n = 2,146)†	188 (18.7)	242 (23.6)	21 (2.1)	11 (1.1)	13 (1.3)	3 (0.3)

Values are n (%). *Data from the FTT Collaborative Group (21). †Includes patients with both LBBB and RBBB.
BBB = bundle branch block; RBBB = right bundle branch block. Other abbreviations as in Table 1.

The 1996 and 2004 guidelines for STEMI were based on the pooled analysis of nine fibinolytic trials which stated that those patients with suspected coronary syndrome presenting with bundle branch block on admission had higher mortality rate and would show maximum decline in mortality rate if started on early fibinolytics.This lended more statistical strength to the patient with bundle branch block and thus formed the basis for formulation for the guidelines for new or presumably new onset LBBB to be treated as STEMI equivalents.

The greatest limitation for this study is that they didn't discriminate between type and chronicity of bundle branch block.Another limitation is that these early fibrinolytic trials were based on less reliable enzymatic markers[CKMB] which can be raised in other non ischemic conditions also.

Hackel DB,Wagner G Ratliff NB et al made an important observation that the new onset LBBB if its due to MI,the the most proximal artery will be involved with greater the magnitude of myocardium involved and the patient would be in acute pulmonary edema or would be in Shock.

With the advent of angiography the new trials categoricaly proved left bundle branch didnt predict the increased risk of AMI. Larson et al study claimed 44% of the patient with new LBBB on subsequent angiogram didn't have culprit occluded artery.Chang et al study found no difference in incidence of MI between new onset and old and with no LBBB [7.3% vs 5.2% vs 6.1%]

Newer 2013 guidelines has taken into consideration all these factors and has removed the new LBBB as an indication for reperfusion therapy.It categorically states that new onset LBBB is not diagnostic of AMI by itself

METHODOLOGY

Study Centre

Coimbatore Medical College Hospital, Coimbatore

Study Period

July 2014 – June 2015

Sample Size

50

Inclusion Criteria

a. Patients of any gender above or equal to 30 years of age at the time of hospital admission
b. Patients presenting with typical anginal pain and suspected Acute coronary syndrome
c. Ecg shows Left Bundle Branch Block

Exclusion Criteria

a. Patients below 30 years of age
b. Patients in acute Heart Failure
c. Hemodynamically unstable patient

Study Design

Short-term prospective study

Method of Data Collection

A total of of fifty(n = 50) patients who were hospitalized for suspected acute coronary syndrome as well as satisfied our inclusion and exclusion criteria were selected for the study.

Data collected included Demographics,History,ECG, and cardiac markers -Troponin T.

Electrocardiograms were classified according to standard guidelines as Left Bundle Branch Block not known to be old[new or presumably new onset] and LBBB known to be old.

Smoker is defined as one who has smoked within the previous one year irrespective of duration of smoking.

Acute Heart failure is defined as the occurrence of acute decompensation of heart function for the first time in patient's life.

A patient is said to be Hemodynamically unstable if the systolic blood pressure is below 90 mm hg or a decrease in mean blood pressure by 30 mmhg

A patient is said to be hypertensive if he/she is already on antihypertensives, and/or if he/she has a high blood pressure documented in the past, and/or if there are signs of long standing hypertension in fundus, ECG, chest X-ray and echocardiogram. JNC VIII guidelines are followed for diagnosing systemic hypertension [74].

A patient is said to be diabetic if he/she is already on oral hypoglycemic agents or insulin therapy, and/or if he/she has a high random/fasting blood sugar value or has a high HBA1c value or has an abnormal oral glucose tolerance test documented in the past, and/or if he/she has elevated blood sugar values during hospital stay. The 2014 guidelines of American Diabetic Association are followed for diagnosing diabetes mellitus.

A patient is said to have coronary artery disease if he/she is already on antiplatelet drugs and nitrates, and/or if he/she has coronary artery disease documented in the past, and/or if he/she has signs of new onset and/or old coronary artery disease in ECG, Echocardiogram and/or percutaneous coronary angiogram.

A patient is said to have dyslipidemia if he/she is already on anti-dyslipidemic drugs such as statins, and/or if he/she has high lipid profile values documented in the past, and/or if he/she is found to have high lipid profile values on admission.

Investigations

Blood sugar

Lipid profile

Chest X-ray PA view

12-lead electrocardiogram

Cardiac Markers – Troponin– T

Transthoracic echocardiogram

The above investigations were done in all patients. Other laboratory and imaging investigations, if needed, were done on a case-by-case basis in selected patients.

Statistical analysis:

Multivariate linear regression analysis with 95% confidence interval (CI) will be done for statistical analysis. Data are expressed in mean ± SE (Standard Error). *P* value <0.05 will be taken as statistically significant. All these analysis will be performed by using a commercially available software Statistical Package for the Social Sciences (SPSS) on personal computer

RESULTS AND OBSERVATIONS

Age in relation to occurrence of new onset and old LBBB

		LBBB		Total
		NEW	OLD	
Age	MORE THAN 50	17	26	43
	LESS THAN 50	6	1	7
Total		**23**	**27**	**50**

P=0.023 significant

Table 4 : Age in relation to occurrence of new onset and old LBBB

73.9% of people with new lbbb are more than 50 years of age 96.3% of people with old lbbb are more than 50 year of age. Older age has higher history of old lbbb which shows as age progress incidence of lbbb is high

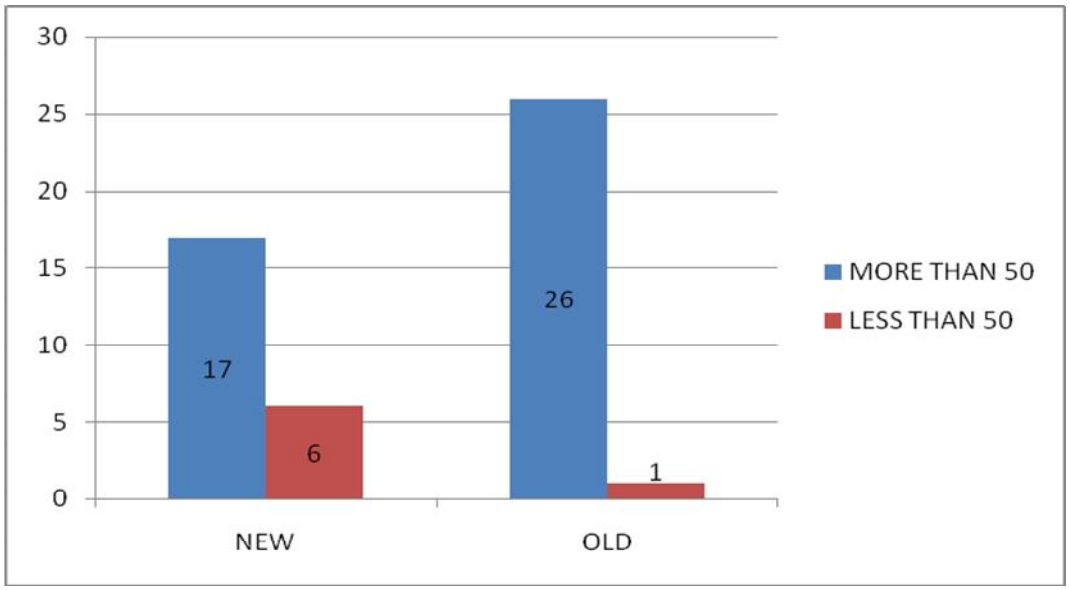


CHART 1 : Age in relation to occurrence of new onset and old LBBB

Gender in relation to new onset and old LBBB

Sex		LBBB		Total
		New	Old	
Male		18	17	35
Female		5	10	15
Total	**23**	**27**	**50**	**Total**

Table 5: Gender in relation to new onset and old LBBB

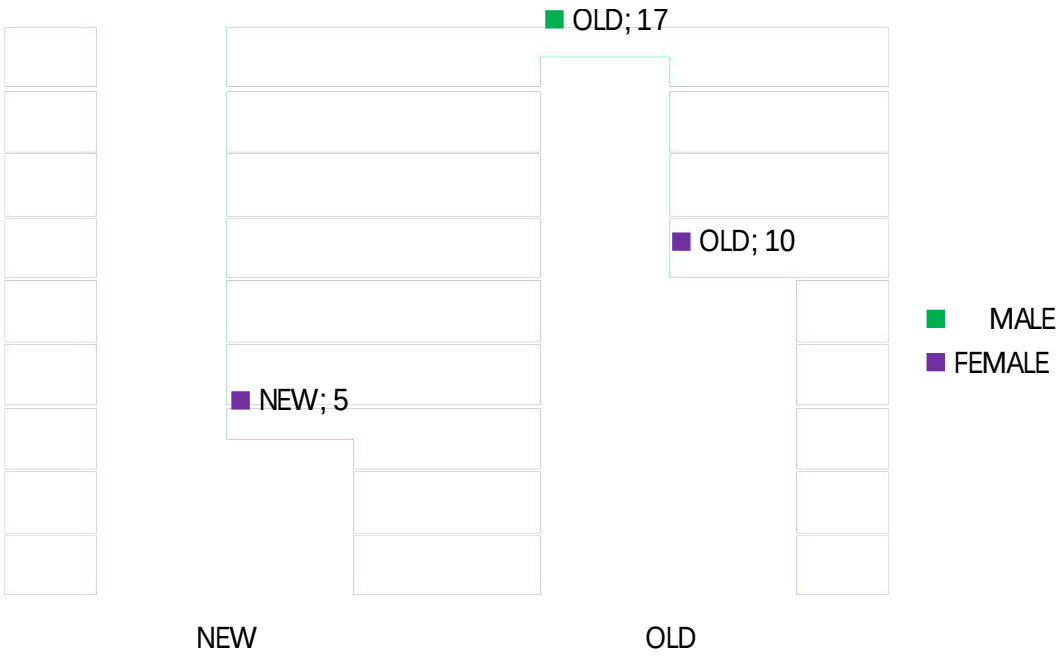


CHART 2 : Gender in relation to new onset and old LBBB

78.3% of patients with new LBBB were males whereas 62.96% of patients with old LBBB were males

TROPONIN CORRELATION IN OLD AND NEW ONSET LBBB IN SUSPECTED ACUTE CORONARY SYNDROME

		LBBB		Total
		NEW	OLD	
TROPT	(+)	2	2	4
	(-)	25	21	46
Total		27	23	50

TABLE 5 : Troponin Correlation In Old And New Onset LBBB In Suspected Acute Coronary Syndrome

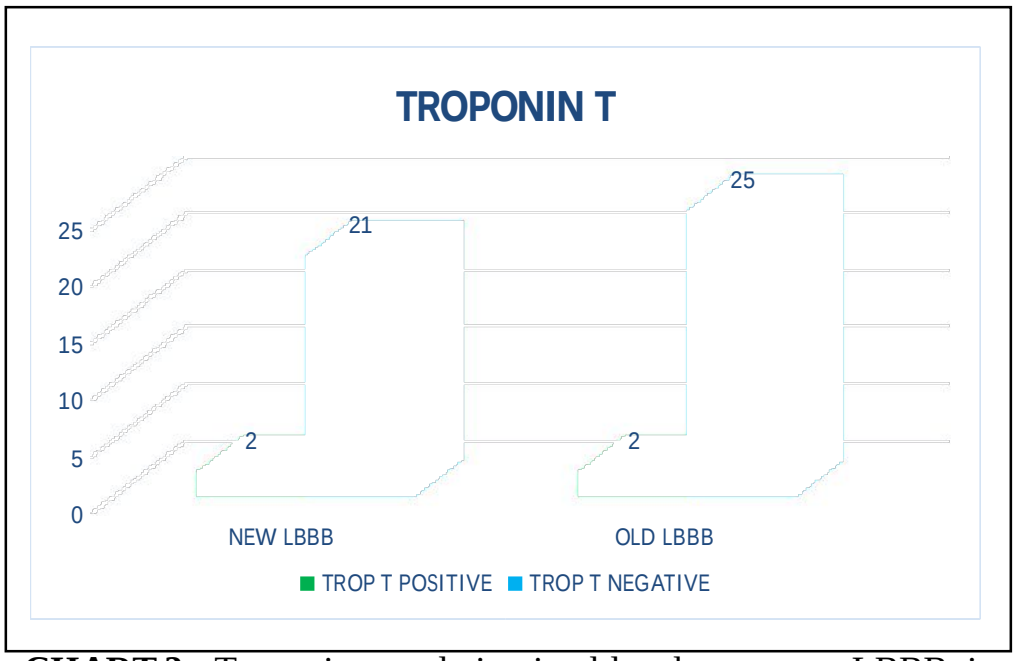


CHART 3 : Troponin correlation in old and new onset LBBB in suspected acute coronary syndrome

P=0.867
OR=0.840

8.7% of the people with new LBBB were troponin positive while 7.4% of the patient with old LBBB had Troponin positivityNo Significant Difference In Incidence Of MI in Both New onset And Old LBBB in suspected Acute coronary syndrome

DIABETES MELLITUS ASSOCIATION IN NEW ONSET AND OLD LBBB IN SUSPECTED ACUTE CORONARY SYNDROME

		LBBB		Total
		NEW	OLD	
	DIABETIC	16	17	33
	NON DIABETIC	7	10	17
Total		**23**	**27**	**50**

Table 6 : Diabetes mellitus association in new onset and old LBBB in suspected acute coronary syndrome

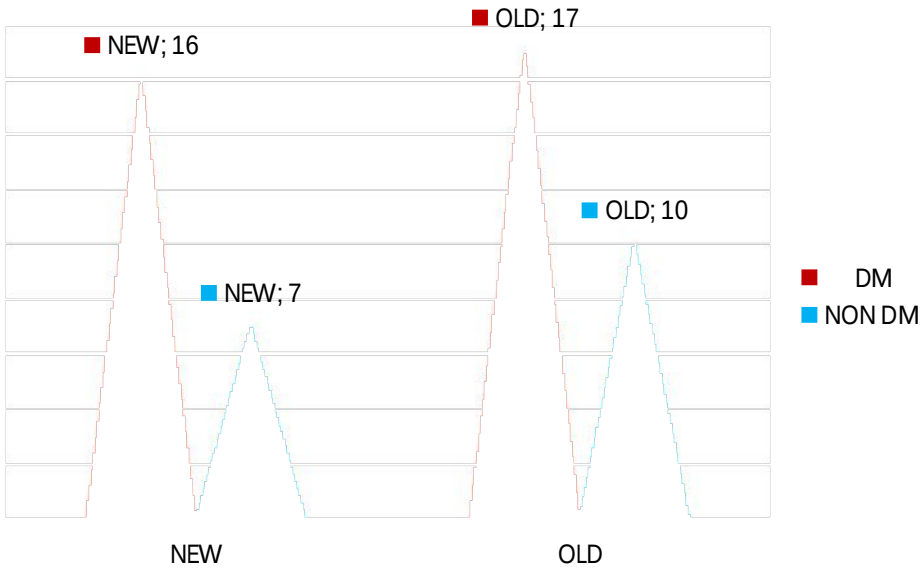


CHART 4 : diabetes mellitus association in new onset and old LBBB in suspected acute coronary syndrome

69.56% OF PATIENTS WITH NEW LBBB HAD DM WHEREAS 62.96% WITH OLD LBBB HAD DM

PRIOR CABG / PTCA ASSOCIATION IN NEW ONSET AND OLD LBBB IN SUSPECTED ACUTE CORONARY SYNDROME

		LBBB		Total
		NEW	OLD	
PRIOR CABG/PTCA	Yes	3	4	7
	No	20	23	43
Total		**23**	**27**	**50**

Table 7 : Prior CABG / PTCA association in new onset and old LBBB in suspected acute coronary syndrome

P = 0.857

OR = 0.863

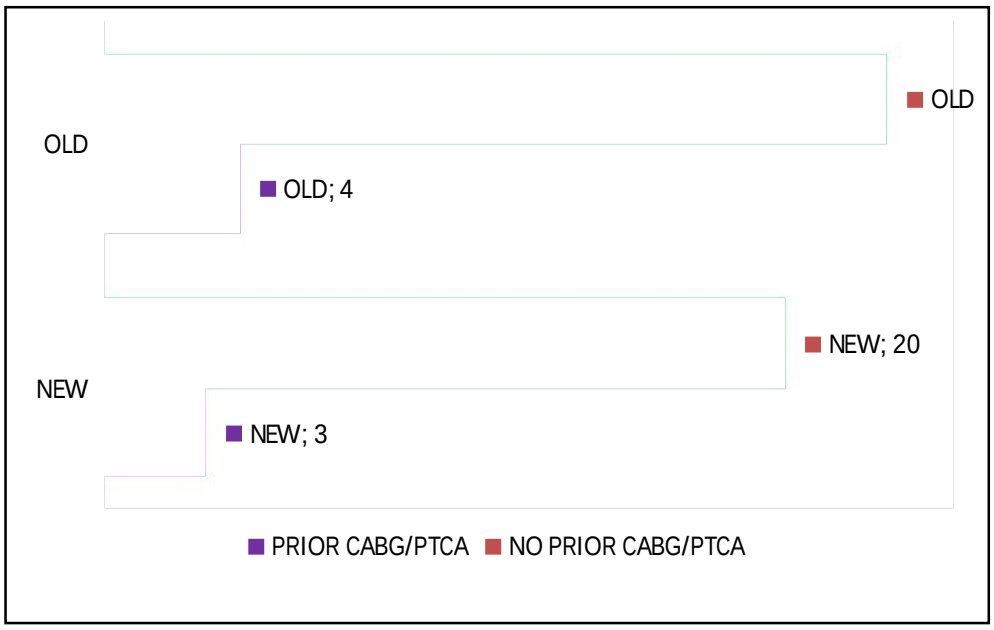


CHART 5 : Prior CABG / PTCA association in new onset and old LBBB in suspected acute coronary syndrome

13% Of Patients With New LBBB Had Prior History Of CABG/PTCA Whereas 14.81% Of With Old LBBB Had Prior History Of CABG/PTCA

PRIOR MI ASSOCIATION IN NEW AND OLD LBBB IN A SUSPECTED ACUTE CORONARY SYNDROME

		LBBB		Total
		NEW	OLD	
Prior MI	Yes	5	8	13
	No	18	19	37
Total		23	27	50

Table 8 : Prior MI association in New and Old LBBB in a suspected acute coronary syndrome

P = 0.526

OR = 0.660

21.73% of patients with new LBBB had prior history OF MI whereas 29.62% of with old LBBB had prior history of MI

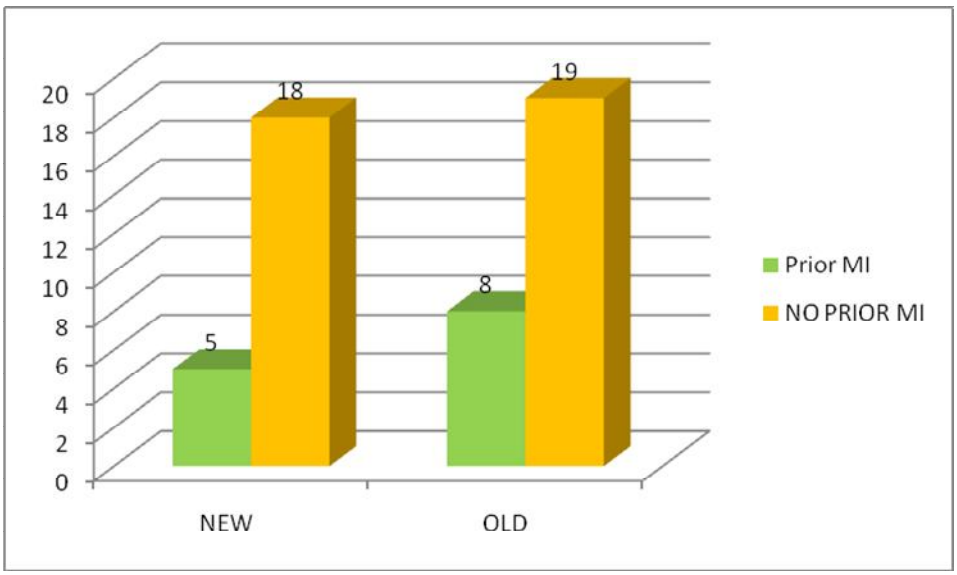


Chart 6 : Prior MI association in New and Old LBBB in a suspected acute coronary syndrome

CAHD ASSOCIATION IN NEW ONSET AND OLD LBBB IN SUSPECTED ACUTE CORONARY SYNDROME

		NEW LBBB	OLD LBBB	
CAHD	Yes	13	17	30
	No	10	10	20
Total		23	27	50

Table 9 : CAHD association in new onset and old LBBB in suspected acute coronary Syndrome

P = 0.643

OR = 0.765

56.52% of patients with new LBBB had prior history of CAHD whereas 62.96% of with old LBBB had prior history of CAHD

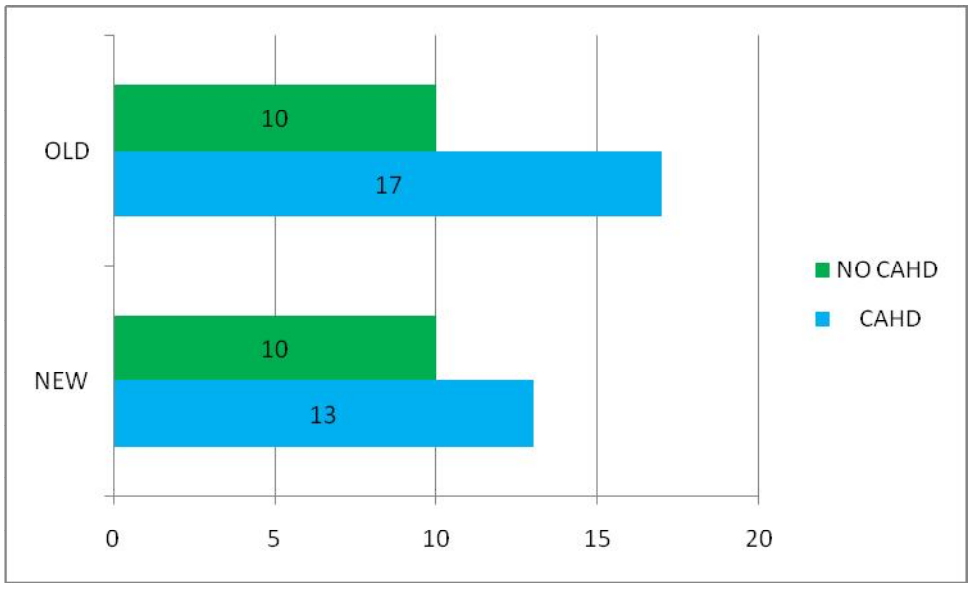


Chart 7 : CAHD ASSOCIATION IN NEW ONSET AND OLD LBBB IN SUSPECTED ACUTE CORONARY SYNDROME

VALVULAR HEART DISEASE ASSOCIATION IN NEW ONSET AND OLD LBBB IN SUSPECTED ACUTE CORONARY SYNDROME

		LBBB		Total
		NEW	OLD	
Valvular	1	2	2	4
	2	21	25	46
Total		23	27	50

Table 10 : valvular heart disease association in new onset and old LBBB in suspected acute coronary syndrome

P = 0.867

OR = 1.190

8.69% of patients with new LBBB had history of Valvular heart disease whereas 7.4% of with old LBBB had prior history of Valvular heart disease

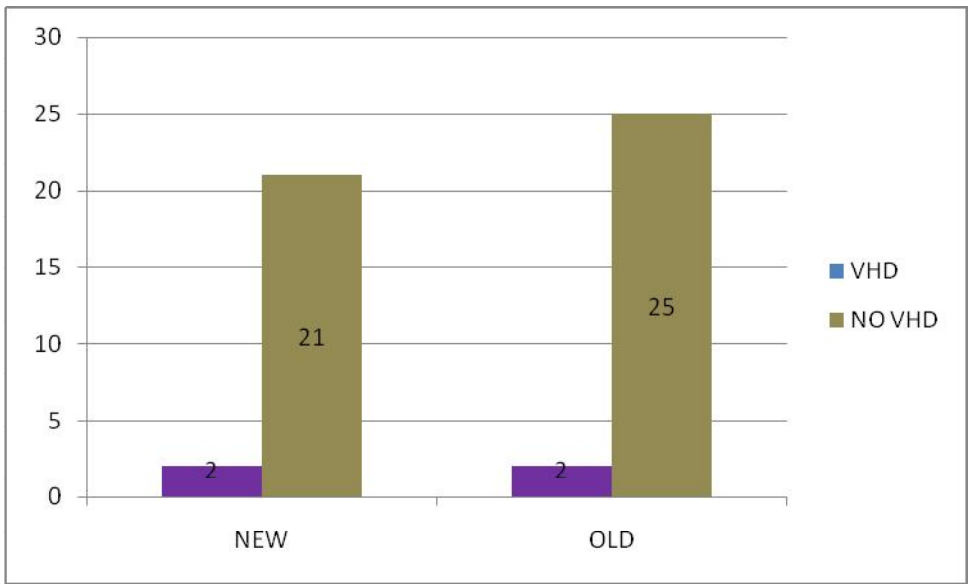


Chart 8 : Valvular heart disease association in new onset and old LBBB in suspected acute coronary syndrome

SYSTEMIC HYPERTENSION ASSOCIATION IN NEW ONSET AND OLD LBBB IN SUSPECTED ACUTE CORONARY SYNDROME

		LBBB		Total
		NEW	OLD	
SHT	1	17	21	38
	2	6	6	12
Total		23	27	50

Table 11: Systemic hypertension association in new onset and old LBBB in suspected acute coronary syndrome

P = 0.750

OR = 0.810

73.91% of patients with new LBBB had prior history of hypertension whereas 77.77%% of with old LBBB had prior history of hypertension

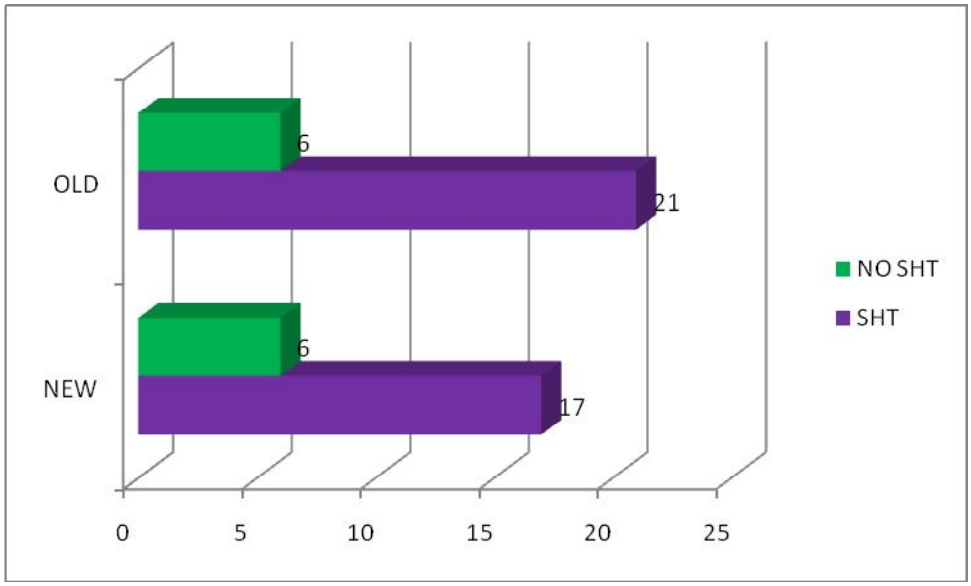


Chart 9 : Systemic hypertension association in new onset and old LBBB in suspected acute coronary syndrome.

HYPERLIPIDEMIA ASSOCIATION IN NEW ONSET AND OLD LBBB IN SUSPECTED ACUTE CORONARY SYNDROME

		LBBB		Total
		NEW	OLD	
Hyperlipidemia	Yes	8	10	18
	No	15	17	32
Total		23	27	50

Table 12 : Hyperlipidemia association in new onset and old LBBB in suspected acute coronary syndrome

P = 0.869

OR = 0.907

Hyperlipidemia doesn't have significant influence on LBBB among this study group there is no significant difference two groups on relation between hyperlipidemia and LBBB

34.78% of patients with new LBBB had prior history of hyperlipidemia whereas 37.03%% of with old LBBB hadprior history of hyperlipidemia

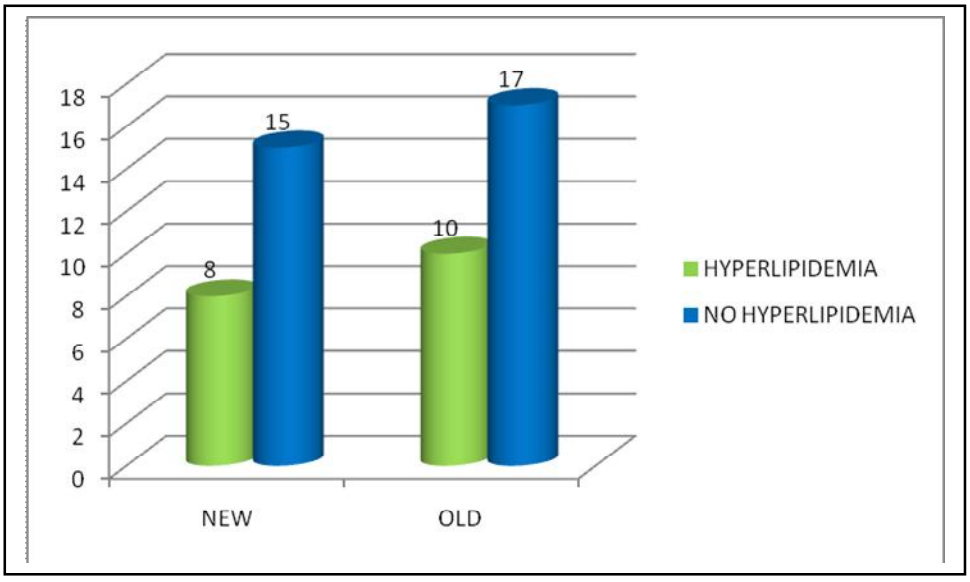


CHART 10 : Hyperlipidemia association in new onset and old LBBB in suspected acute coronary syndrome

SMOKING ASSOCIATION IN NEW ONSET AND OLD LBBB IN SUSPECTED ACUTE CORONARY SYNDROME

		LBBB		Total
		NEW	OLD	
Smoker	Yes	16	17	33
	No	7	10	17
Total		23	27	50

Table 13 : Smoking association in new onset and old LBBB in suspected acute coronary syndrome

P = 0.623

OR = 1.345

69.56% of patients with new LBBB had were smokers whereas 62.96% of with old LBBB were smokers

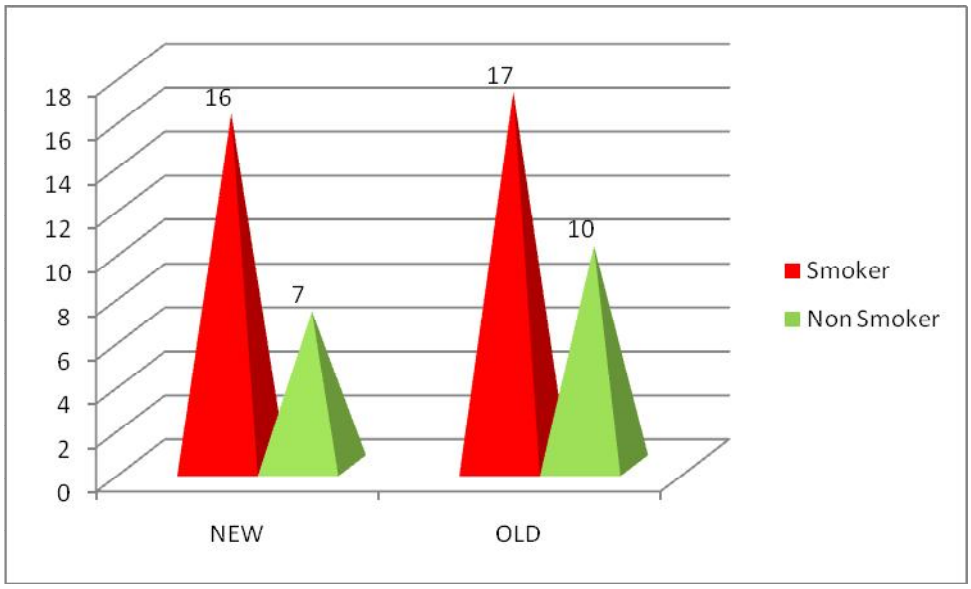


CHART 11 : Smoking association in new onset and old LBBB in suspected acute coronary syndrome

FAMILY HISTORY OF MI ASSOCIATION WITH NEW AND OLD LBBB IN A SUSPECTED ACUTE CORONARY SYNDROME.

		LBBB		Total
		NEW	OLD	
Family History of MI	Yes	7	10	17
	No	16	17	33
Total		**23**	**27**	**50**

Table 14 : Family history of MI association with new and old LBBB in a suspected Acute Coronary Syndrome

P = 0.623

OR = 0.744

30.43% of patients with new LBBB had family history of CAHD whereas 37.03% of patients with old LBBB had family history of CAHD

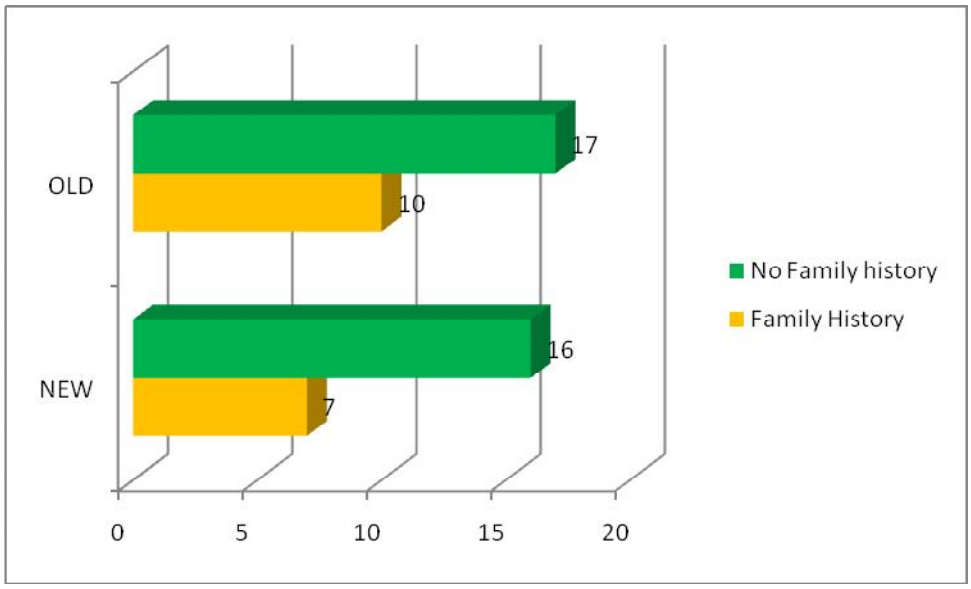


Chart 12 : Family history of MI association with new and old LBBB in a suspected Acute Coronary Syndrome

TIMI SCORE ASSOCIATION WITH NEW AND OLD LBBB IN A SUSPECTED ACUTE CORONARY SYNDROME

		LBBB		Total
		NEW	OLD	
TIMI SCORE	0-1	7	7	14
	1-3	11	14	25
	>-4	5	6	11
Total		23	27	50

Table 15 : TIMI score association with new and old LBBB in a suspected acute coronary syndrome.

P = 0.936

TIMI score doesn't have significant influence on LBBB among this study group there is no significant difference two groups on risk of incidence of mi based on TIMI score

		NEW LBBB	OLD LBBB
TIMI SCORE	0-1	30.43%	25.92%
	2-3	47.82%	51.85%
	>_4	21.73%	22.22%

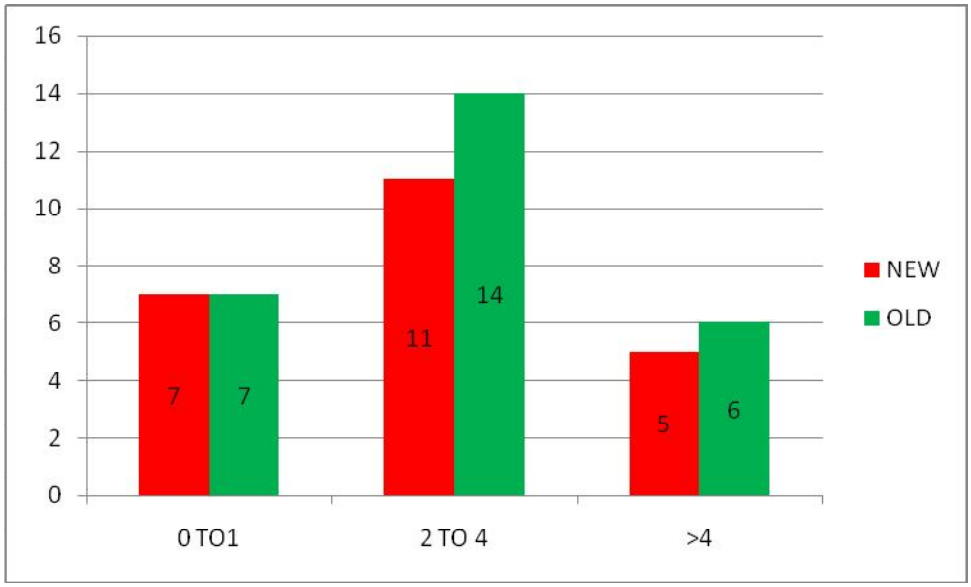


Chart 13 : TIMI score association with new and old LBBB in a suspected acute coronary syndrome.

DISCUSSION

We concluded that new or presumed Left bundle branch need not imply that the patient has a acute myocardial infarction. The previous studies which were the basis for the development of the guidelines before 2013 relied on patients enrolled in large randomized studies. It is therefore quite logical to conclude these patients enrolled in registries are different from actual patients presenting to casualty with acute chest syndrome.

We conclude the incidence of MI in new onset LBBB is not significantly different from those that of people with old LBBB. But many studies predicted worse outcome in AMI with new LBBB than with old LBBB.

Prior guidelines recommend reperfusion for new or presumed new LBBB. But it need not be as our study suggests the rate MI is not different in people with new or old LBBB. Primary PCI is a more reasonable approach because it will aid in both diagnosis and also in intervention if any is needed.

New diagnostic methods are the need of the hour for selecting candidates for reperfusion. The need for new strategies are far more greater in centres with out primary PCI facility as the complication in

fibrinolytic therapy is much greater than that of falsely activating cath lab.Potential strategies include

1]Different algorithmic approach for cath lab activation for a Patient with LBBB with acute chest syndrome than than the patient with a ST segment elevation myocardial infarction

2]Use of more focused ,specified ECG criterias [Sgarbossa,Modified Smith criteria]

3]Wide spread use of cardiac bio markers

4]Use of bed side echo

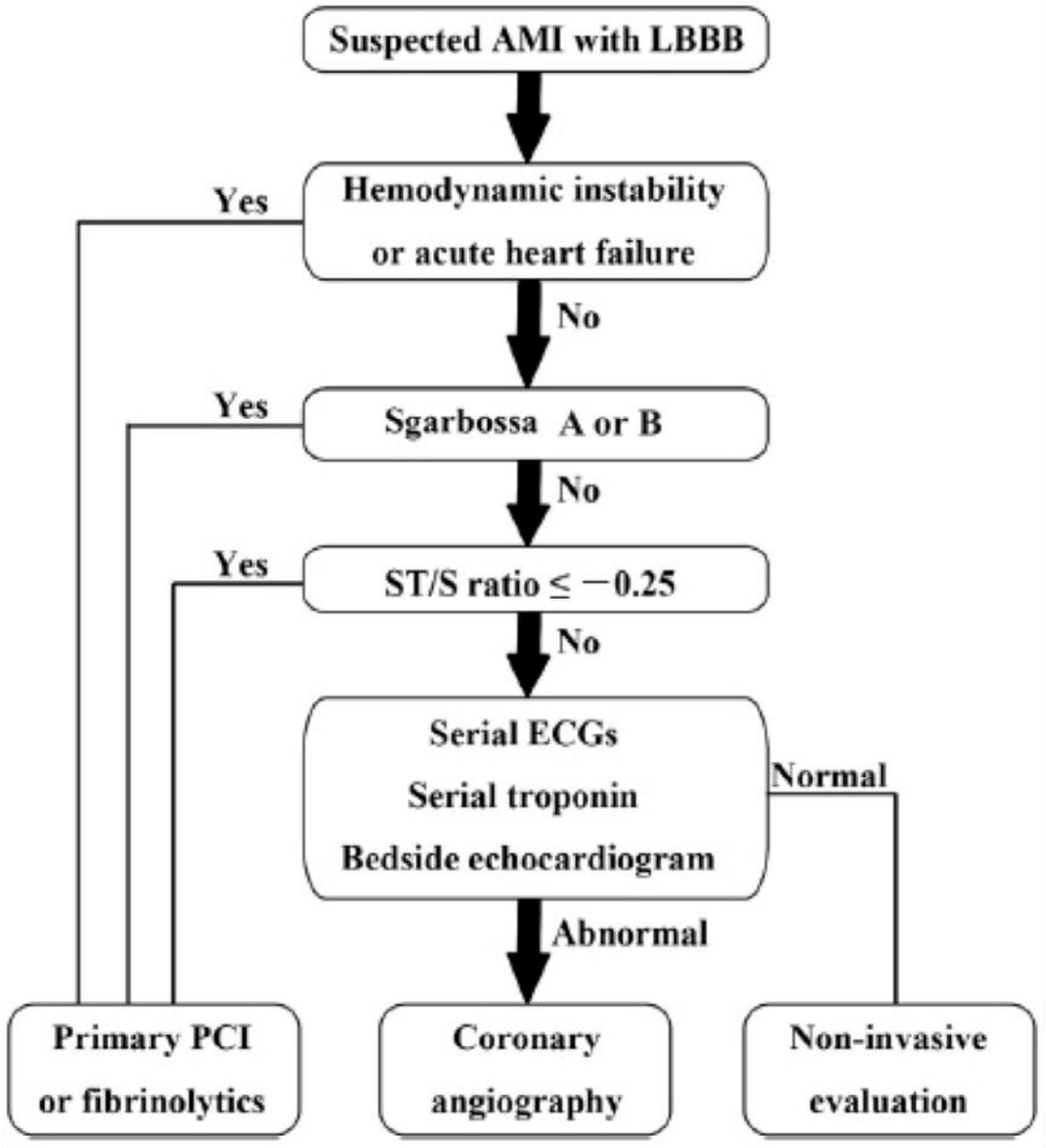
If the time interval between first medical contact and balloon is less than 120 minutes PCI is far superior to fibrinolysis given the dreaded complications of fibrinolysis.If the interval exceed 2 hours onsite fibrinolysis is beneficial.But this may not be applicable to patients with LBBB because many of them dont have MI and LBBB itself has inherent risk of bleeding[with preexistent heart disease,hypertension,heart failure]For these patients PCI is the preferred strategy and fibrinolysis should be considered with utmost caution if the likelihood of MI is very high!

Testing cardiac biomarkers, specifically the cardiac troponins I and T, is promising in the diagnosis of AMI with LBBB. In recent years,

the analytic sensitivity for troponin detection has improved hundredfold . Newer assays have improved sensitivity as well, which enables 2 troponin titres with a minute difference of few picograms per milliliter to be reliably differentiated. This is significant because mere rise in cardiac troponin can be found in many chronic cardiac and noncardiac conditions, and thus not specific AMI, a fast rise in absolute levels of troponin strongly supports the diagnosis of an evolving AMI . A serial fast rise in troponin in a patient with LBBB, especially in the background of ongoing chest discomfort, may denote a hidden STEMI and may prompt further testing such as bedside echocardiography ,primary PCI , or fibrinolytic therapy if PCI is not available.

In contrast, a more slow rise and lower peak in troponin levels may signal an NSTEMI (in which case, transfer to a PCI-capable facility still would be recommended typically), whereas a static troponin level would suggest a non-ACS cause. Although there are limited datas, it is necessary to hasten the timing of enzyme assessment, so that measurements are performed every 15 min, rather than every 60 to 90 min, in patients with LBBB and suspected AMI. Such an approach would shorten the delay in reperfusion in those ultimately determined to have STEMI equivalents. Assessment of serial rapid biomarker measuremen , should be the focus of additional study in patients with suspected AMI and LBBB.

Table : 17 Algorithmic approach for patients with suspected Acute Coronary Syndrome and new onset LBBB



A new diagnostic and triage algorithm has been proposed considering the new guideline recommendations for a suspected acute coronary syndrome patient in the setting of new onset LBBB.If the patient is in cardiogenic shock or in acute heart failure ,Myocardial Infarction is

strongly suspected and early reperfusion should be promptly started.Sgarbossa criteria has been extremely helpful in detecting myocardial Infarction because of its reduced false positive rates and high specificity.If the sgarbossa score is 3 the patient can be confidently considered acute MI and reperfusion therapy can be started.If the score is less than two,then the smiths criteria shold be used anf if the ST/S ratio is less than or equal to 0.25 then the patient is a candidate for reperfusion.If none of the criteria is met ,Patient should be evaluated further with serial ECG monitoring,serial troponin levels and bed side echocardiography.If there is ST segment changes in serial electrocardiograms or rapid rise in troponin levels or there is any wall motion abnormalities without chronic changes in echocardiography,decision for cath lab should be promptly decided .

SUMMARY

Patient Characteristics	New onset LBBB	Old LBBB
Age{>50}	73.9%	96.3%
Sex[Male]	78.3%	62.96%
Cardiac Risk Factors		
Hypertension	73.91%	77.77%
Diabetes	69.56%	62.96%
Hyperlipidemia	34.78%	37.03%
Family History of CAD	30.43%	37.03%
Smoker	60.56%	62.96%
Medical History		
H/O CAD	56.52%	62.96%
PRIOR MI	21.73%	29.62%

PRIOR CABG/PTCA		13%	14.81%
TIMI SCORE	0-1	30.43%	25.92%
	2-3	47.82%	51.85%
	>_4	21.73%	22.22%

73.9% of people with new LBBB are more than 50 years of age 96.3% of people with old lbbb are more than 50 year of age. Older age has higher history of old LBBB which shows as age progress incidence of lbbb is high

8.7% of the people with new LBBB were troponin positive while 7.4% of the patient with old LBBB had Troponin positivity.Thus there is no Significant Difference in the incidence of MI in both new onset and Old LBBB in suspected Acute coronary syndrome.

CONCLUSION

There is no difference in the incidence of Myocardial Infarction in the patients with old LBBB and new onset LBBB.

Hemodynamically stable Patients with suspected Acute Coronary Syndrome and new onset LBBB are not at increased risk of AMI.

Presence of LBBB whether new or old LBBB did not predict acute myocardial Infarction

Algorithmic approach towards a suspected Acute coronary syndrome should take into consideration hemodynamic status ,whether patient is in acute heart failure AND whether ECG fits into Smiths criteria and if any of them is present ,decision for reperfusion should be promptly taken.

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PROFORMA

Serial No.:

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Address:

IP. No.:

D.O.A:

D.O.D:

Heart rate on admission:

Systolic blood pressureon admission:

ECG on admission:Old or new LBBB

Prior MI: Yes/No

Prior CAHD: Yes/No

Prior CABG/PTCA: Yes/No

Famiy History of CAHD: Yes/No

Smoking: Yes/No

SHT: Yes/No

DM: Yes/No

Dyslipidemia: Yes/No

Troponin T:Positive/Negative

Echo for wall motion abnormalities:Present/Absent

MASTER CHART ABBREVIATIONS

BP	–	Blood pressure
PR	-	Pulse rate
Ecg	–	Electrocardiogram
LBBB	–	Left Bundle Branch Block
Echo	-	Echocardiogram
DM	-	Diabetes Mellitus
CABG	-	Coronary Artery Bypass grafting
MI	-	Myocardial Infarction
CAHD	-	Coronary Artery Heart Disease
TIMI Score	-	Thrombolysis in Myocardial Infarction Score

CONSENT FORM

I am **Dr .ASHOK S**, carrying out a study on the topic **"UTILITY OF LEFT BUNDLE BRANCH BLOCK AS THE DIAGNOSTIC CRITERION FOR MYOCARDIAL INFARCTION IN A HEMODYNAMICALLY STABLE PATIENT".**

My research project is being carried out under the Department of General Medicine, Coimbatore Medical College and Hospital, Coimbatore.

RESEARCH BEING DONE:

"**UTILITY OF LEFT BUNDLE BRANCH BLOCK AS THE DIAGNOSTIC CRITERION FOR MYOCARDIAL INFARCTION IN A HEMODYNAMICALLY STABLE PATIENT"**

SAMPLE SIZE:

50 patients.

STUDY PARTICIPANTS:

Patients attending General Medicine Out Patient Department with the complaints of chest pain , Coimbatore Medical College and Hospital, Coimbatore.

LOCATION:

Coimbatore Medical College and Hospital, Coimbatore.

You, Shri./ Smt./ Kum. _____, aged ____ years, S/o / D/o / W/o _____, residing at _____ _____ are requested to be a participant in the research study **titled "UTILITY OF LEFT BUNDLE BRANCH BLOCK AS THE DIAGNOSTIC CRITERION FOR MYOCARDIAL INFARCTION IN A HEMODYNAMICALLY STABLE PATIENT"** in Government Medical College Hospital, Coimbatore. You satisfy eligibility criteria as per the inclusion criteria. You can ask any question or seek any clarifications on the study that you may have before agreeing to participate.

DECLINE FROM PARTICIPATION

You are hereby made aware that participation in this study is purely voluntary and honorary and that you have the option and the right to decline from participation in the study.

PRIVACY AND CONFIDENTIALITY

You are hereby assured about your privacy. Privacy of subject will be respected and any information about you or provided by you during the study will be kept strictly confidential.

AUTHORIZATION TO PUBLISH RESULTS

Results of the study may be published for scientific purposes and/or presented to scientific groups, however you will not be identified; neither will your privacy be breached.

STATEMENT OF CONSENT

I, _____, do hereby volunteer and consent to participate in this study being conducted by **Dr.ASHOKS ..** I have read and understood the consent form / or it has been read and explained to me in my own language. The study has been fully explained to me, and I may ask questions at any time.

Signature / Left Thumb Impression of the Volunteer Date: Place:

Signature and Name of witness Date: Place:

Signature of the investigator:

Name of the investigator:

xggpj;go tk;

bgah; :

ghypdk; :

Kfthp : taJ :

muR nfhit kUj;Jtf;fy;Yhpa;y; bghJ kUj;Jt Jiwapy; gl;l nkwgogg[gapYk;khz th;**kU.R. mnrhf;**mth;fs; "**nfhaKj;Jh; kUj;Jt fy;Yhp kUj;Jtkidapy; khuilg;ig fzL mwpjypy; ,lJ flL ff;pis milggpd; gadghL**" Ma;tpy; nkw;nfhs;Sk; bra;Kiw kw;Wk; midj;J tputu';fisak;nflL f;bfhzL vdJ ren;jf';fis bjspthLj;jpf; bfhz;nld; vdgij bjhptpj;Jf; bfhs;fpnwd;

ehd;,ej Ma;tpy;KG rk;kjj;Jld;/Ra rpej ida[lDk;fye;J bfhs;s rk;kjpf;fpnwd;

,ej Ma;tpy; vd;Dila midj;J tpgu';fs; ghJfhf;fggLtJld; ,jd; Kotpfs; Ma;tpjHpy; btspapLggLtjpy; Mlnrgid ,y;iy vdgij bjhptpj;Jf bfhs;fpnwd; vej neuj;jpy; mej Ma;tpypUe;J ehd; tpyf;f bfhs;s vdf;F chpik czL vdgijak;mwpntd;

,lk;: ifbahggk;/nuif

ehs;:

MASTER CHART

Name	Age	Sex	BP	PI	Trop T	ECG	Echo	DM	PRIOR C	Prior MI	CAH	Valvular Heart Disease	Systemic Hypertension	Hyperlip	Smoker
						LBBB									
Marimuthu	**58**	**M**	**12O/80**	**74**	**N**	**Old**	**2**	**Y**	**Y**	**Y**	**Y**	**N**	**Y**	**Y**	**Y**
karupusamy	63	M	110/82	78	N	New	2	Y	N	N	Y	N	Y	Y	Y
chinammal	**49**	**F**	**120/70**	**88**	**N**	**New**	**2**	**N**	**Y**	**Y**	**Y**	**N**	**Y**	**Y**	**N**
Kannan	78	M	126/84	86	N	Old	2	Y	N	N	Y	N	Y	N	Y
isakimuthu	48	M	110/80	88	N	Old	2	Y	N	N	N	N	Y	Y	Y
ramasamy	90	M	116/78	94	N	Old	2	Y	N	Y	Y	N	N	N	N
Sekar	38	M	138/90	70	N	New	2	N	N	N	N	Y	Y	N	Y
Chandran	57	M	120/80	78	N	New	2	N	N	Y	N	N	N	N	Y
Alagappan	**48**	**M**	**126/84**	**68**	**P**	**New**	**1**	**Y**	**N**	**N**	**Y**	**N**	**Y**	**N**	**Y**
kaliyammal	76	F	148/94	82	N	New	2	Y	N	N	Y	N	Y	N	N
Asokan	52	M	126/76	84	N	Old	2	Y	N	N	N	Y	Y	Y	Y
Periyanayagi	**56**	**F**	**110/70**	**98**	**P**	**Old**	**1**	**N**	**Y**	**Y**	**Y**	**N**	**Y**	**N**	**N**
Govindaraj	64	M	128/80	74	N	Old	2	N	N	N	N	N	Y	N	Y
Muthu	31	M	120/80	78	N	New	2	Y	N	N	N	N	Y	Y	Y
kalaiarasan	66	M	138/84	82	N	Old	2	N	Y	Y	Y	N	Y	N	Y
Senthil	54	M	120/80	76	N	Old	2	Y	N	N	Y	N	N	N	Y
Radha	60	F	140/78	72	P	Old	2	Y	N	N	N	N	Y	N	N
Manivannam	42	M	130/86	78	N	New	2	N	N	N	N	N	Y	N	Y
Gopi	**65**	**M**	**120/80**	**86**	**P**	**New**	**1**	**Y**	**Y**	**Y**	**N**	**N**	**Y**	**Y**	**N**
Devaraj	60	M	128/88	82	N	New	2	N	N	N	N	N	N	N	Y
Sudalaiappan	53	M	130/80	88	N	Old	2	Y	N	N	N	N	Y	N	Y
Ramathal	65	F	140/90	90	N	Old	2	N	N	Y	Y	N	Y	Y	N
Sanmugam	**70**	**M**	**124/86**	**74**	**P**	**Old**	**1**	**N**	**N**	**N**	**Y**	**Y**	**Y**	**Y**	**Y**
Rakayi	85	F	120/80	76	N	Old	2	Y	N	N	Y	N	Y	Y	N
sathyan	76	M	160/90	84	N	Old	2	N	N	Y	Y	N	Y	N	Y
vimala	84	F	124/78	88	N	Old	2	Y	N	N	Y	N	Y	N	N
Kalaivani	56	F	134/88	82	N	Old	2	N	N	N	N	N	Y	Y	Y
Chinasamy	74	M	120/80	90	N	Old	2	Y	N	Y	Y	N	Y	N	N
rajasekar	66	M	100/56	94	N	New	2	Y	N	Y	Y	N	Y	Y	Y
giridharan	55	M	136/70	76	N	Old	2	N	N	N	N	N	Y	N	Y
Kandan	65	M	134/80	66	N	Old	2	Y	N	N	N	N	Y	N	Y
veerasamy	54	M	138/84	74	N	Old	2	N	N	N	Y	N	Y	Y	Y
rekha	26	F	134/82	72	N	New	2	N	N	N	N	Y	N	N	N
ramanadan	64	M	124/84	78	N	Old	2	Y	Y	Y	Y	N	N	N	Y

senathipadi	72	M	136/76	84	N	New	2	Y	N	N	Y	N	Y	N	Y
vinodini	53	F	126/84	88	N	Old	2	N	N	N	N	N	N	N	N
veerammal	61	F	110/66	90	P	Old	2	Y	N	N	Y	N	Y	N	N
arjunasamy	65	M	140/90	92	N	New	2	N	N	N	Y	N	Y	N	N
govindan	66	M	124/84	88	N	New	2	Y	N	N	Y	N	Y	N	Y
Murugan	72	M	128/76	70	N	Old	2	Y	N	N	Y	N	Y	Y	Y
kupanan	68	M	130/80	74	N	New	2	Y	N	N	Y	N	Y	N	Y
sabeena	56	F	122/86	78	N	Old	2	Y	N	N	N	N	N	Y	N
kataboman	54	F	136/84	80	N	New	2	Y	Y	Y	Y	N	Y	N	N
krishnammal	67	F	120/80	84	N	New	2	Y	N	N	N	N	Y	Y	N
kumaran	64	M	128/88	76	N	New	2	Y	N	N	Y	N	N	Y	Y
karupusamy	58	M	130/86	78	N	New	2	Y	N	N	Y	N	Y	N	Y
vedammal	67	F	128/86	74	N	Old	1	Y	N	N	Y	N	N	N	Y
visakan	75	M	120/86	78	N	New	2	Y	N	N	Y	N	Y	Y	Y
anandan	50	M	130/80	82	N	New	2	Y	N	N	N	N	N	N	Y
kumaraguru	65	M	110/66	76	N	New	2	Y	N	N	N	N	N	N	Y